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Your SELECT statement is:
s gabp

Items	File
148	5: Biosis Previews(R)_1969-2002/Apr W4
153	34: SciSearch(R) Cited Ref Sci_1990-2002/May W1
18	35: Dissertation Abs Online_1861-2002/Apr
10	65: Inside Conferences_1993-2002/Apr W4
77	71: ELSEVIER BIOBASE_1994-2002/May W1
101	73: EMBASE_1974-2002/Apr W4
6	77: Conference Papers Index_1973-2002/Mar
2	94: JICST-EPlus_1985-2002/Mar W3
9	98: General Sci Abs/Full-Text_1984-2002/Mar
19	144: Pascal_1973-2002/May W1
5	149: TGG Health&Wellness DB(SM)_1976-2002/Apr W3
131	155: MEDLINE(R)_1966-2002/Apr W4
29	156: ToxFile_1966-2002/Feb W4
69	159: Cancerlit_1975-2002/Mar
1	162: CAB HEALTH_1983-2002/Mar
1	172: EMBASE Alert_2002/May W1
5	266: FEDRIP_2002/Mar
3	370: Science_1996-1999/Jul W3
123	399: CA SEARCH(R)_1967-2002/UD=13619

SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2002/Apr W4
(c) 2002 BIOSIS
File 34:SciSearch(R) Cited Ref Sci 1990-2002/May W1
(c) 2002 Inst for Sci Info
File 73:EMBASE 1974-2002/Apr W4
(c) 2002 Elsevier Science B.V.
*File 73: For information about Explode feature please
see Help News73.
File 155:MEDLINE(R) 1966-2002/Apr W4
*File 155: This file will be reloaded. Accession numbers will change.

?s gabp
S7 533 GABP
?s s7 and (p300 or cbp)
533 S7
12027 P300
5609 CBP

S8 4 S7 AND (P300 OR CBP)
?rd
...completed examining records
S9 1 RD (unique items)

9/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11900137 BIOSIS NO.: 199900146246
GA-binding protein factors, in concert with the coactivator CREB binding protein/ p300 , control the induction of the interleukin 16 promoter in T lymphocytes.
AUTHOR: Bannert Norbert; Avots Andris; Baier Michael(a); Serfling Edgar; Kurth Reinhard
AUTHOR ADDRESS: (a)Paul-Ehrlich-Inst., Paul-Ehrlich-Strasse 51-59, D-63225 Langen**Germany
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 96 (4):p1541-1546 Feb. 16, 1999
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Interleukin 16 (IL-16) is a chemotactic cytokine that binds to the CD4 receptor and affects the activation of T cells and replication of HIV. It is expressed as a large 67-kDa precursor protein (pro-IL-16) in lymphocytes, macrophages, and mast cells, as well as in airway epithelial cells from asthmatics after challenge with allergen. This pro-IL-16 is subsequently processed to the mature cytokine of 13 kDa. To study the expression of IL-16 at the transcriptional level, we cloned the human chromosomal IL-16 gene and analyzed its promoter. The human IL-16 gene consists of seven exons and six introns. The 5' sequences up to nucleotide -120 of the human and murine IL-16 genes share >84% sequence homology and harbor promoter elements for constitutive and inducible transcription in T cells. Although both promoters lack any TATA box, they contain two CAAT box-like motifs and three binding sites of GA-binding protein (GABP) transcription factors. Two of these motifs are part of a highly conserved and inducible dyad symmetry element shown previously to control a remote IL-2 enhancer and the CD18 promoter. In concert with the coactivator CREB binding protein/ p300 , which interacts with GABPalpha, the binding of GABPalpha and -beta to the dyad symmetry element controls the induction of IL-16 promoter in T cells. Supplementing the data on the processing of pro-IL-16, our results indicate the complexity of IL-16 expression, which is tightly controlled at the transcriptional and posttranslational levels in T lymphocytes.

DESCRIPTORS:
MAJOR CONCEPTS: Immune System (Chemical Coordination and Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae); murine (Muridae)
ORGANISMS: PARTS ETC: T lymphocytes--blood and lymphatics, immune system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: interleukin 16--expression, promoter; interleukin 2; CREB binding protein/ p300 ; GA-binding protein
MISCELLANEOUS TERMS: nucleotide sequence
CONCEPT CODES:
34502 Immunology and Immunochemistry-General; Methods
02502 Cytology and Cytochemistry-General
03502 Genetics and Cytogenetics-General
10060 Biochemical Studies-General
15001 Blood, Blood-Forming Organs and Body Fluids-General; Methods
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

R.R. Genuario et al., "Comparative utilization of transcription factor GABP by the promoters of ribosomal protein genes rpL30 and rpL32," Gene Expression, 3(3):279-288 (1993)
Abstract.

Set Items Description
S1 552 GABP OR HGABP
S2 0 S1 AND PHOSPHORYL$
S3 45 S1 AND PHOSPHORYL?
S4 15 RD (unique items)
4/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13525968 BIOSIS NO.: 200200154789
Neuregulin-1-stimulated phosphorylation of GABP .
AUTHOR: Fromm Larry(a); Burden Steven J
AUTHOR ADDRESS: (a)Skirball Institute, NYU Medical Center, 540 First Avenue, New York, NY, 10016**USA
JOURNAL: Molecular Biology of the Cell 11 (Supplement):p288a Dec., 2000

MEDIUM: print
CONFERENCE/MEETING: 40th American Society for Cell Biology Annual Meeting San Francisco, CA, USA December 09-13, 2000
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Molecular Genetics (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
CHEMICALS & BIOCHEMICALS: Erk; GABP -alpha; GABP -beta; Jnk; acetylcholine receptor; neuregulin-1
GENE NAME: AChR gene--transcription
MISCELLANEOUS TERMS: muscle cell; neuromuscular synapse; Meeting Abstract
CONCEPT CODES:
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
02502 Cytology and Cytochemistry-General
02506 Cytology and Cytochemistry-Animal
03502 Genetics and Cytogenetics-General
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
17504 Muscle-Physiology and Biochemistry

4/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13482747 BIOSIS NO.: 200200111568
An ERK2 docking site in the Pointed domain distinguishes a subset of ETS transcription factors.
AUTHOR: Seidel Jeffrey J; Graves Barbara J(a)
AUTHOR ADDRESS: (a)Huntsman Cancer Institute, Department of Oncological Sciences, University of Utah, Salt Lake City, UT, 84112-5550**USA E-Mail: barbara.graves@hci.utah.edu
JOURNAL: Genes & Development 16 (1):p127-137 January 1, 2002
MEDIUM: print
ISSN: 0890-9369
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The ETS transcription factors perform distinct biological functions despite conserving a highly similar DNA-binding domain. One distinguishing property of a subset of ETS proteins is a conserved region of 80 amino acids termed the Pointed (PNT) domain. Using enzyme kinetics

we determined that the Ets-1 PNT domain contains an ERK2 docking site. The docking site enhances the efficiency of phosphorylation of a mitogen-activated protein kinase (MAPK) site N-terminal to the PNT domain. The site enhances ERK2 binding rather than catalysis. Three hydrophobic residues are involved in docking, and the previously determined NMR structure indicates that these residues are clustered on the surface of the Ets-1 PNT domain. The docking site function is conserved in the PNT domain of the highly related Ets-2 but not in the ets family member GABPalpha. Ablation of the docking site in Ets-1 and Ets-2 prevented Ras pathway-mediated enhancement of the transactivation function of these proteins. This study provides structural insight into the function of a MAPK docking site and describes a unique activity for the PNT domain among a subset of ets family members.

REGISTRY NUMBERS: 142243-02-5: MITOGEN-ACTIVATED PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics)
CHEMICALS & BIOCHEMICALS: ETS transcription factors--ERK2 docking site, Pointed domain; Ets-1 protein; Ets-2 protein; GABP -alpha; Ras protein; mitogen-activated protein kinase-- phosphorylation efficiency
METHODS & EQUIPMENT: NMR--analytical method
MISCELLANEOUS TERMS: enzyme kinetics
CONCEPT CODES:
10802 Enzymes-General and Comparative Studies; Coenzymes

4/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13054181 BIOSIS NO.: 200100261330
Neuregulin-1-stimulated phosphorylation of GABP in skeletal muscle cells.
AUTHOR: Fromm Larry; Burden Steven J(a)
AUTHOR ADDRESS: (a)Molecular Neurobiology Program, Skirball Institute of Biomolecular Medicine, NYU Medical School, 540 First Avenue, New York, NY, 10016: burden@saturn.med.nyu.edu**USA
JOURNAL: Biochemistry 40 (17):p5306-5312 May 1, 2001
MEDIUM: print
ISSN: 0006-2960
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Localization of acetylcholine receptors (AChRs) to neuromuscular synapses is mediated, in part, through selective transcription of AChR genes in myofiber synaptic nuclei. Neuregulin-1 (NRG-1) is a good candidate for the extracellular signal that induces synapse-specific gene expression, since NRG-1 is concentrated at synaptic sites and activates AChR synthesis in cultured muscle cells. NRG-1-induced transcription requires activation of Erk and Jnk MAP kinases, but the downstream substrates that mediate this transcriptional response are not known. Previous studies have demonstrated that a consensus binding site for Ets proteins is required both for NRG-1-induced transcription and for synapse-specific transcription in transgenic mice. This regulatory element binds GABPalpha, an Ets protein, and GABPbeta, a protein that dimerizes with GABPalpha, raising the possibility that phosphorylation of GABP by MAP kinases induces transcription of AChR genes. To determine whether MAP kinases might directly regulate the activity of GABP , we studied MAP kinase-catalyzed and NRG-1-induced phosphorylation of GABPalpha and GABPbeta. We show that GABPalpha and GABPbeta are phosphorylated in vitro by Erk and by Jnk. Using recombinant proteins containing mutated serine and threonine resides, we show that GABPalpha is phosphorylated predominantly at threonine 280, while serine 170 and threonine 180 are the major phosphorylation sites in GABPbeta. We generated antibodies specific to the major phosphorylation site in GABPalpha and show that NRG-1 stimulates phosphorylation of GABPalpha at threonine 280 in vivo. These results suggest that GABPalpha is a target of MAP kinases in NRG-1-stimulated muscle cells and are consistent with the idea that phosphorylation of GABPalpha contributes to transcriptional activation of AChR genes by NRG-1.
REGISTRY NUMBERS: 142243-02-5: EXTRACELLULAR SIGNAL-REGULATED KINASE;
155215-87-5: C-JUN N-TERMINAL KINASE; 142243-02-5: MAP KINASE;
142243-02-5: MITOGEN-ACTIVATED PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics); Molecular Genetics (Biochemistry and Molecular Biophysics); Muscular System (Movement and Support)
ORGANISMS: PARTS ETC: neuromuscular synapse--muscular system, nervous system; skeletal muscle cells--muscular system
CHEMICALS & BIOCHEMICALS: Erk {extracellular signal-regulated kinase}; GABP -alpha--amino acid mutation, phosphorylation , transcription factor complex; GABP -beta--amino acid mutation, phosphorylation , transcription factor complex; Jnk {C-jun N-terminal kinase}; MAP kinase {mitogen-activated protein kinase}; acetylcholine receptors { AChRs}--transcriptional activation; monoclonal antibodies; neuregulin-1 {NRG-1
CONCEPT CODES:
10802 Enzymes-General and Comparative Studies; Coenzymes

02506 Cytology and Cytochemistry-Animal
03502 Genetics and Cytogenetics-General
17504 Muscle-Physiology and Biochemistry
20504 Nervous System-Physiology and Biochemistry
34502 Immunology and Immunochemistry-General; Methods

4/9/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12455986 BIOSIS NO.: 200000209488
Role of GABPalpha/GABPbeta in neuregulin signaling in muscle.
AUTHOR: Fromm L(a); Burden S J(a)
AUTHOR ADDRESS: (a)Skirball Institute, NYU Medical Center, New York, NY, 10016**USA
JOURNAL: Society for Neuroscience Abstracts 25 (1-2):p1557 1999
CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA October 23-28, 1999
SPONSOR: Society for Neuroscience
ISSN: 0190-5295
RECORD TYPE: Citation
LANGUAGE: English
SUMMARY LANGUAGE: English
REGISTRY NUMBERS: 191809-35-5: NEUREGULIN
DESCRIPTORS:
MAJOR CONCEPTS: Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination)
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: mouse (Muridae)--transgenic
ORGANISMS: PARTS ETC: acetylcholine receptors--localization; muscle cells--cultured, muscular system; neuromuscular synapse--nervous system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: GABP -alpha protein-- phosphorylation ; GABP -beta protein-- phosphorylation ; neuregulin; neuregulin-response element--mutation; mouse AChR-delta subunit-hGH gene (Muridae)--fusion gene
MISCELLANEOUS TERMS: signal transduction; synapse-specific gene expression; Meeting Abstract
CONCEPT CODES:
03506 Genetics and Cytogenetics-Animal
17504 Muscle-Physiology and Biochemistry

20504 Nervous System-Physiology and Biochemistry
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
BIOSYSTEMATIC CODES:
86375 Muridae

4/9/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11537322 BIOSIS NO.: 199800318654
Implication of a multisubunit Ets-related transcription factor in synaptic expression of the nicotinic acetylcholine receptor.
AUTHOR: Schaeffer Laurent; Duclert Nathalie; Huchet-Dymanus Monique; Changeux Jean-Pierre(a)
AUTHOR ADDRESS: (a)CNRS UA D1284 'Neurobiologie Moleculaire', Inst. Pasteur, 25 rue du Dr Roux, F-75724 Paris Cedex**France
JOURNAL: EMBO (European Molecular Biology Organization) Journal 17 (11):p 3078-3090 June 1, 1998
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In adult muscle, transcription of the nicotinic acetylcholine receptor (AChR) is restricted to the nuclei located at the neuromuscular junction. The N-box, a new promoter element, was identified recently and shown to contribute to this compartmentalized synaptic expression of the AChR delta- and epsilon-subunits. We demonstrate that the N-box mediates transcriptional activation in cultured myotubes and identify the transcription factor that binds to the N-box as a heterooligomer in myotubes and adult muscle. The GABP (GA-binding protein) alpha-subunit belongs to the Ets family of transcription factors, whereas the beta-subunit shares homology with IkappaB and Drosophila Notch protein. GABP binding specificity to mutated N-box in vitro strictly parallels the sequence requirement for beta-galactosidase targeting to the endplate in vivo. In situ hybridization studies reveal that the mRNAs of both GABP subunits are abundant in mouse diaphragm, with preferential expression of the alpha-subunit at motor endplates. In addition, heregulin increases GABPalpha protein levels and regulates phosphorylation of both subunits in cultured chick myotubes. Finally, dominant-negative mutants of either GABPalpha or GABPbeta block heregulin-elicited transcriptional activation of the AChR delta and epsilon genes. These findings establish the expected connection with a presynaptic trophic factor whose release contributes to the accumulation

of AChR subunit mRNAs at the motor endplate.

REGISTRY NUMBERS: 51-84-3: ACETYLCHOLINE; 191809-35-5D: NEUREGULINS;
9026-43-1: PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination)
BIOSYSTEMATIC NAMES: Galliformes--Aves, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: chicken (Galliformes); mouse (Muridae)
ORGANISMS: PARTS ETC: diaphragm--muscular system; motor endplate--nervous system; muscle--muscular system; myotubes--muscular system; synapse
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Birds; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: multisubunit Ets-related transcription factor ; mRNA {messenger RNA}--expression; neuregulins; nicotinic acetylcholine receptor delta-subunit gene--expression; nicotinic acetylcholine receptor epsilon-subunit gene--expression; Drosophila Notch protein; GA-binding protein alpha subunit; I-kappa-B--transcription factor
METHODS & EQUIPMENT: immunoprecipitation--analytical method, methodological approach; in situ hybridization--analytical method, methodological approach; metabolic labeling--analytical method, methodological approach; mitogen-activated protein kinase assay--analytical method, methodological approach; mobility shift assay--analytical method, methodological approach; mobility supershift assay --analytical method, methodological approach; Northern blot--analytical method, methodological approach
MISCELLANEOUS TERMS: synaptic gene expression; transcriptional activation; N-box
CONCEPT CODES:
03506 Genetics and Cytogenetics-Animal
10060 Biochemical Studies-General
17501 Muscle-General; Methods
20501 Nervous System-General; Methods
BIOSYSTEMATIC CODES:
85536 Galliformes
86375 Muridae

4/9/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11476274 BIOSIS NO.: 199800257606
The GABP -responsive element of the interleukin-2 enhancer is regulated by JNK/SAPK-activating pathways in T lymphocytes.
AUTHOR: Hoffmeyer Angelika; Avots Andris; Flory Egbert; Weber Christoph K; Serfling Edgar; Rapp Ulf R(a)
AUTHOR ADDRESS: (a)Inst. Med. Strahlenkunde Zellforschung, Univ. Wuerzburg, Versbacher Strasse 5, D-97078 Wuerzburg**Germany
JOURNAL: Journal of Biological Chemistry 273 (17):p10112-10119 April 24, 1998
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T cell activation leads via multiple intracellular signaling pathways to rapid induction of interleukin-2 (IL-2) expression, which can be mimicked by costimulation with 12-O-tetradecanoylphorbol-13-acetate (TPA) and ionomycin. We have identified a distal IL-2 enhancer regulated by the Raf-MEK-ERK signaling pathway, which can be induced by TPA/ionomycin treatment. It contains a dyad symmetry element (DSE) controlled by the Ets-like transcription factor GA-binding protein (GABP), a target of activated ERK. TPA/ionomycin treatment of T cells stimulates both mitogen-activated ERK as well as the stress-activated mitogenactivated protein kinase family members JNK/SAPK and p38. In this study, we investigated the contribution of the stress-activated pathways to the induction of the distal IL-2 enhancer. We show that JNK- but not p38-activating pathways regulate the DSE activity. Furthermore, the JNK/SAPK signaling pathway cooperates with the Raf-MEK-ERK cascade in TPA/ionomycin-induced DSE activity. In T cells, overexpression of SPRK/MLK3 an activator of JNK/SAPK strongly induces DSE-dependent transcription and dominant negative kinases of SEK and SAPK impair TPA/ionomycin-induced DSE activity. Blocking both ERK and JNK/SAPK pathways abolishes the DSE induction. The inducibility of the DSE is strongly dependent on the Ets-core motifs, which are bound by GABP . Both subunits of GABP are phosphorylated upon JNK activation in vivo and three different isoforms of JNK/SAPK, but not p38, in vitro. Our data suggest that GABP is targeted by signaling events from both ERR and JNK/SAPK pathways. GABP therefore is a candidate for signal integration and regulation of IL-2 transcription in T lymphocytes.

REGISTRY NUMBERS: 9031-44-1: KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Immune System (Chemical Coordination and Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata,

Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: A3.01 (Hominidae)--human T lymphoma cells; HEK293 (Hominidae) ; NIH-3T3 (Muridae)
ORGANISMS: PARTS ETC: T lymphocyte--activation, blood and lymphatics, immune system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans;
Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: interleukin-2 enhancer--GA-binding protein-responsive element regulation, characterization; tpa; DSE--induction; GA-binding protein { GABP }--transcription factor; SPRJ/MLK3--JNK/SAPK activator, overexpression
METHODS & EQUIPMENT: bacterially expressed protein purification--methodological approach, purification method; electrophoretic mobility shift assay--analysis/characterization techniques, electrophoretic techniques, analytical method; immunoblotting--analytical method, methodological approach; immunoprecipitation--precipitation techniques , purification method; kinase assay--analytical method, methodological approach; transient transfection metabolic labeling and reporter gene assays--methodological approach; DNA cloning--methodological approach
MISCELLANEOUS TERMS: intracellular signalling; transcription regulation ; JNK/SAPK-activating pathways
CONCEPT CODES:
34508 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
02506 Cytology and Cytochemistry-Animal
02508 Cytology and Cytochemistry-Human
03506 Genetics and Cytogenetics-Animal
03508 Genetics and Cytogenetics-Human
10300 Replication, Transcription, Translation
10506 Biophysics-Molecular Properties and Macromolecules
10808 Enzymes-Physiological Studies
13012 Metabolism-Proteins, Peptides and Amino Acids
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
17002 Endocrine System-General
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

4/9/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11448644 BIOSIS NO.: 199800229976
Regulation of mitochondrial biogenesis in brown adipose tissue: Nuclear respiratory factor-2/GA-binding protein is responsible for the transcriptional regulation of the gene for the mitochondrial ATP synthase beta subunit.
AUTHOR: Villena Josep A; Vinas Octavi; Mampel Teresa; Iglesias Roser; Giralt Marta; Villarroya Francesc(a)
AUTHOR ADDRESS: (a)Dep. Biochem. Mol. Biol., Univ. Barcelona, Diagonal 645, 08028 Barcelona**Spain
JOURNAL: Biochemical Journal 331 (1):p121-127 April, 1998
ISSN: 0264-6021
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The regulation of transcription of the gene for the beta subunit of the FoF1 ATP synthase (ATPsynbeta) in brown adipose tissue has been studied as a model to determine the molecular mechanisms for mitochondrial biogenesis associated with brown adipocyte differentiation. The expression of the ATPsynbeta mRNA is induced during the brown adipocyte differentiation that occurs during murine prenatal development or when brown adipocytes differentiate in culture. This induction occurs in parallel with enhanced gene expression for other nuclear and mitochondrially-encoded components of the respiratory chain/oxidative phosphorylation system (OXPHOS). Transient transfection assays indicated that the expression of the ATPsynbeta gene promoter is higher in differentiated HIB-1B brown adipocytes than in non-differentiated HIB-1B cells. A major transcriptional regulatory site was identified between nt - 306 and - 266 in the ATPsynbeta promoter. This element has a higher enhancer capacity in differentiated brown adipocyte HIB-1B cells than in non-differentiated cells. Electrophoretic shift analysis indicated that Sp1 and nuclear respiratory factor-2/GA-binding protein (NRF2/ GABP) were the main nuclear proteins present in brown adipose tissue that bind this site. Double-point mutant analysis indicated a major role for the NRF2/ GABP site in the enhancer capacity of this element in brown fat cells. It is proposed that NRF2/ GABP plays a pivotal role in the coordinated enhancement of OXPHOS gene expression associated with mitochondrial biogenesis in brown adipocyte differentiation.

REGISTRY NUMBERS: 37205-63-3: ATP SYNTHASE
DESCRIPTORS:
MAJOR CONCEPTS: Bioenergetics (Biochemistry and Molecular Biophysics); Enzymology (Biochemistry and Molecular Biophysics); Skeletal System (Movement and Support)
ORGANISMS: PARTS ETC: brown adipose tissue--skeletal system

CHEMICALS & BIOCHEMICALS: nuclear respiratory factor-2/GA-binding protein; ATP synthase beta-subunit--mitochondrial
MISCELLANEOUS TERMS: mitochondrial biogenesis regulation; thermogenesis ; transcriptional gene regulation
CONCEPT CODES:
13003 Metabolism-Energy and Respiratory Metabolism
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10300 Replication, Transcription, Translation
10510 Biophysics-Bioenergetics: Electron Transport and Oxidative Phosphorylation
10806 Enzymes-Chemical and Physical
18004 Bones, Joints, Fasciae, Connective and Adipose Tissue-Physiology and Biochemistry

4/9/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11069780 BIOSIS NO.: 199799690925
GABP factors bind to a distal interleukin 2 (IL-2) enhancer and contribute to c-Raf-mediated increase in IL-2 induction.
AUTHOR: Avots Andris; Hoffmeyer Angelika; Flory Egbert; Cimanis Alexander; Rapp Ulf R; Serfling Edgar(a)
AUTHOR ADDRESS: (a)Inst. Pathol., Josef-Schneider-Str. 2, D-97080 Wuerzburg **Germany
JOURNAL: Molecular and Cellular Biology 17 (8):p4381-4389 1997
ISSN: 0270-7306
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Triggering of the T-cell receptor-CD3 complex activates two major signal cascades in T lymphocytes, (i) Ca-2+-dependent signal cascades and (ii) protein kinase cascades. Both signal cascades contribute to the induction of the interleukin 2 (IL-2) gene during T-cell activation. Prominent protein kinase cascades are those that activate mitogen-activated protein (MAP) kinases. We show here that c-Raf, which is at the helm of the classic MAP-Erk cascade, contributes to IL-2 induction through a distal enhancer element spanning the nucleotides from positions -502 to -413 in front of the transcriptional start site of the IL-2 gene. Induction of this distal IL-2 enhancer differs from induction of the proximal IL-2 promoter-enhancer, since it is induced by phorbol esters alone and independent from Ca-2+ signals. In DNA-protein binding studies, we detected the binding of transcription factors GABP -alpha and -beta to a dyad symmetry element (DSE) of the distal enhancer, which

is formed by palindromic binding sites of Ets-like factors. Introduction of point mutations suppressing GABP binding to the DSE interfered with the induction of the distal enhancer and the entire IL-2 promoterenhancer, while overexpression of both GABP factors enhanced the IL-2 promoter-enhancer induction. Overexpression of BXB, a constitutive active version of c-Raf, and of further members of the Ras-Raf-Erk signal cascade exerted an increase of GABP -mediated promoter-enhancer induction. In conjunction with previously published data on c-Raf-induced phosphorylation of GABP factors (E. Flory, A. Hoffmeyer, U. Smola, U. R. Rapp, and J. T. Bruder, J. Virol. 70:2260-2268, 1996), these results indicate a contribution of GABP factors to the Raf-mediated enhancement of IL-2 induction during T-cell activation.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Endocrine System (Chemical Coordination and Homeostasis); Genetics
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: JURKAT (Hominidae)--cell line
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; humans; mammals; primates; vertebrates
MISCELLANEOUS TERMS: Research Article; A 3.01 CELL LINE; BINDING; BLOOD AND LYMPHATICS; EXPRESSION; GABP FACTOR; GENE EXPRESSION; HUMAN T LEUKEMIA; HUMAN T LYMPHOMA; IMMUNE SYSTEM; INTERLEUKIN-2; INTERLEUKIN-2 ENHANCER; INTERLEUKIN-2 GENE; MOLECULAR GENETICS; T-CELL; TRANSCRIPTION FACTOR
CONCEPT CODES:
03508 Genetics and Cytogenetics-Human
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
17002 Endocrine System-General
15006 Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
24010 Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
BIOSYSTEMATIC CODES:
86215 Hominidae

4/9/9 (Item 9 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

10922183 BIOSIS NO.: 199799543328
GA-binding protein is involved in altered expression of ribosomal protein L32 gene.
AUTHOR: Curcic Dusica; Glibetic Marija; Larson Dawn E; Sells Bruce H(a)
AUTHOR ADDRESS: (a)Dep. Molecular Biol. Genetics, Univ. Guelph, Guelph, ON N1G 2W1**Canada
JOURNAL: Journal of Cellular Biochemistry 65 (3):p287-307 1997
ISSN: 0730-2312
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Differentiation of BC-3H1 myoblasts to myocytes is accompanied by a 67% drop in the rate of rpL32 gene transcription. Addition of high concentrations of serum to resting myocyte populations stimulates cell growth and subsequent dedifferentiation to proliferating myoblasts with a return to the normal rate of rpL32 gene transcription. During these growth rate changes the binding activities of previously identified factors (beta, gamma, delta) which interact with the rpL32 gene promoter were examined by mobility shift assays. Binding of the beta factor (an Ets related protein) to an oligonucleotide containing the beta element was reduced significantly in myocyte nuclear extracts, but subsequent dedifferentiation increased binding within 30 min in either the presence or absence of the cycloheximide. Binding of the gamma and delta factors to their respective elements changed only slightly during these processes. Dephosphorylation of either myoblast or myocyte extracts resulted in increased binding of the beta factor suggesting that binding activity of the beta factor is modulated by phosphorylation during the changes in BC-3H1 myoblasts growth rate. In addition, mobility shift assays with recombinant GABP alpha and beta proteins and their specific antibodies revealed that GABP proteins bind to the rpL32 gene promoter in a sequence dependent manner, and that similar proteins are present in BC3H1 myoblast/myocyte extracts. These results support the premise that the GABP heterodimer is the rpL32 beta factor. Furthermore, during BC-3H1 myoblast differentiation and dedifferentiation neither the levels of the GABP et and beta proteins nor their respective mRNAs change. These results suggest that GABP is a constitutively expressed protein and is involved in regulating rpL32 gene by post-transcriptional modifications.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology; Development; Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics)

BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Muridae (Muridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals;
nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
MISCELLANEOUS TERMS: Research Article; ALPHA-SUBUNIT; BC-3-H1 CELL LINE
; BETA-SUBUNIT; CELL BIOLOGY; DEDIFFERENTIATION; DIFFERENTIATION;
EXPRESSION; GA-BINDING PROTEIN; GROWTH; HETERODIMER; MOLECULAR GENETICS
; MYOBLAST; POST-TRANSCRIPTIONAL REGULATION; PROMOTER; RIBOSOMAL
PROTEIN L32 BETA-FACTOR; RIBOSOMAL PROTEIN L32 GENE
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10300 Replication, Transcription, Translation
25508 Developmental Biology-Embryology-Morphogenesis, General
BIOSYSTEMATIC CODES:
86375 Muridae

4/9/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10625592 BIOSIS NO.: 199699246737
Redox regulation of GA-binding protein-alpha DNA binding activity.
AUTHOR: Martin Mark E(a); Chinenov Yurii; Yu Mi; Schmidt Tonya K; Yang Xiu-Ying
AUTHOR ADDRESS: (a)Dep. Biochem., Univ. Missouri, Columbia, MO 65212**USA
JOURNAL: Journal of Biological Chemistry 271 (41):p25617-25623 1996
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have investigated the reduction/oxidation (redox) regulation of the heteromeric transcription factor GA-binding protein (GABP). GABP , also known as nuclear respiratory factor 2, regulates the expression of nuclear encoded mitochondrial proteins involved in oxidative phosphorylation , including cytochrome c oxidase subunits IV

and Vb, as well as the expression of mitochondrial transcription factor 1. GABP is composed of two subunits, the Ets-related GABP -alpha, which mediates specific DNA binding, and GABP -beta, which forms heterodimers and heterotetramers on DNA sequences containing the PEA3/Ets motif ((C/A)GGA(A/T)(G/A)). We demonstrate here that GABP DNA binding activity and GABP -dependent gene expression in 3T3 cells are inhibited by pro-oxidant conditions. DNA binding of recombinant GABP -alpha was activated by chemical reduction (dithiothreitol) and by thioredoxin; however, GSSG inhibited GABP DNA binding activity. Treatment of GABP -alpha, but not GABP -beta-1, with sulfhydryl-alkylating agents also inhibited GABP DNA binding activity. Our results suggest that GABP DNA binding activity is redox-regulated in vivo, possibly by thioredoxin-mediated reduction and by GSSG-mediated oxidation of the GABP -alpha subunit. The regulation of GABP (nuclear respiratory factor 2) DNA binding activity by cellular redox changes provides an important link between mitochondrial and nuclear gene expression and the redox state of the cell.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Bioenergetics (Biochemistry and Molecular Biophysics); Cell Biology; Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Muridae (Muridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
MISCELLANEOUS TERMS: DNA; GA-BINDING PROTEIN; GABP ; HETEROMERIC TRANSCRIPTION FACTOR; MITOCHONDRIAL REDOX STATE; MOLECULAR GENETICS; NIH 3T3 CELL LINE; NUCLEAR GENE EXPRESSION; REDOX REGULATION
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10300 Replication, Transcription, Translation
10506 Biophysics-Molecular Properties and Macromolecules
10510 Biophysics-Bioenergetics: Electron Transport and Oxidative Phosphorylation
BIOSYSTEMATIC CODES:
86375 Muridae

4/9/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10368811 BIOSIS NO.: 199698823729
GABP mediates insulin-increased prolactin gene transcription.
AUTHOR: Ouyang Liaohan(a); Jacob Kirsten K; Stanley Frederick M
AUTHOR ADDRESS: (a)Mt. Sinai Med. Cent., Asher Levy Place, New York, NY 10029**USA
JOURNAL: Journal of Biological Chemistry 271 (18):p10425-10428 1996
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The insulin-response element from the prolactin gene is identical to the Ets-binding site, and dominant-negative Ets protein inhibits insulin-increased prolactin gene expression. Immunoblotting identified the Ets-related transcription factor GABP in nuclear extracts from GH cells. Expression of GABP -alpha and GABP -beta-1 squelches insulin-increased prolactin gene expression. GABP -alpha and GABP -beta-1 bind the insulin-response element of the prolactin promoter, and anti- GABP -alpha and anti- GABP -beta-1 antibodies supershift a species seen with nuclear extracts from GH cells. GABP -alpha immunoprecipitated from insulin-treated, 32P-labeled GH cells was phosphorylated 3-fold more than GABP -alpha from control cells. There was no increase in phosphorylation of GABP -beta in response to insulin. Mitogen-activated protein (MAP) kinase activity is increased 10-fold in insulin-treated GH4 cells. MAP kinase immunoprecipitated from control cells does not phosphorylate GABP -alpha while MAP kinase immunoprecipitated from insulin-treated cells shows substantial phosphorylation of GABP -alpha. These studies suggest that GABP mediates insulin-increased transcription of the prolactin gene. GABP may be regulated by MAP kinase phosphorylation .

REGISTRY NUMBERS: 9002-62-4: PROLACTIN; 9026-43-1: PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics); Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Muridae (Muridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals;
nonhuman vertebrates; nonhuman mammals; rodents; vertebrates

CHEMICALS & BIOCHEMICALS: PROLACTIN; PROTEIN KINASE
MISCELLANEOUS TERMS: GENE REGULATION; GH4 CELLS; INSULIN-RESPONSE
ELEMENT; MITOGEN-ACTIVATED PROTEIN KINASE; PROMOTER; SIGNAL TRANSDUCTION
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10300 Replication, Transcription, Translation
10808 Enzymes-Physiological Studies
17008 Endocrine System-Pancreas
17014 Endocrine System-Pituitary
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
BIOSYSTEMATIC CODES:
86375 Muridae

4/9/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10320360 BIOSIS NO.: 199698775278
Raf-1 kinase targets GA-binding protein in transcriptional regulation of the human immunodeficiency virus type 1 promoter.
AUTHOR: Flory Egbert; Hoffmeyer Angelika; Smola Ute; Rapp Ulf R(a); Bruder Joseph T
AUTHOR ADDRESS: (a)Inst. Radiobiol. Cell Res., Univ. Wuerzburg, D-97078 Wuerzburg**Germany
JOURNAL: Journal of Virology 70 (4):p2260-2268 1996
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The serine/threonine protein kinase Raf-1 is a component of a conserved intracellular signaling cascade that controls responses to various extracellular stimuli. Transcription from several promoters, including the oncogene-responsive element in the polyomavirus enhancer, the c-fos promoter, as well as other AP-1- and Ets-dependent promoters, can be induced by Raf-1 kinase. Previously, we have shown that activated Raf-I kinase transactivates the human immunodeficiency virus type 1 (HIV-1) long terminal repeat and have identified the NF-kappa-B binding motif as a Raf-1-responsive element (RafRE). We now report that Raf-1 kinaseinduced transactivation from the HIV RafRE involves the purine-rich-repeat-binding protein (GABP), which is composed of two

distinct subunits (alpha- and beta). GABP alpha is an Ets oncogene-related DNA-binding protein, and GABP beta contains four ankyrin-like repeats that have been shown to be essential in protein-protein interactions. In electrophoretic mobility shift assays using nuclear extracts from human Jurkat T cells, a protein-DNA complex which was supershifted with antiserum against GABP alpha and GABP beta was observed. Purified recombinant GABP alpha and beta interact with the HIV RafRE as judged from DNA binding assays. Cotransfection experiments with GABP alpha and beta and Raf-1 kinase demonstrate synergistic transactivation of the HIV-1 promoter. Point mutations in the HIV RafRE abolished the Raf-1 kinase- as well as GABP alpha- and beta-induced transactivation. The observed Raf-1- GABP synergism presumably involves phosphorylation of GABP subunits, phosphorylation of GABP in vivo. However, GABP is not a target of Raf-1 kinase; instead, it is a substrate of mitogen-activated protein kinase (MAPK/ERK), since in vitro phosphorylation of GABP alpha and beta was achieved by the reconstituted protein kinase cascade but not with purified Raf-1 or MEK. These results suggest that Raf-1 kinase-induced activation of the HIV-1 promoter is mediated by the classical cytoplasmic cascade resulting in MAPK/ERK-mediated phosphorylation of GABP alpha and beta. Because the HIV RafRE corresponds to a region within the promoter which is essential for regulation of HIV-1 expression, the data indicate that in addition to NF-kappa-B, GABP transcription factors are important for induced expression of HIV.

REGISTRY NUMBERS: 139691-76-2: RAF-1 KINASE; 9026-43-1: SERINE-THREONINE
PROTEIN KINASE; 9026-43-1: PROTEIN KINASE; 120-73-0: PURINE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Genetics; Metabolism; Microbiology; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Papovaviridae--Viruses; Retroviridae--Viruses
ORGANISMS: Hominidae (Hominidae); Papovaviridae (Papovaviridae); Retroviridae (Retroviridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; humans; mammals; microorganisms; primates; vertebrates; viruses
CHEMICALS & BIOCHEMICALS: RAF-1 KINASE; SERINE-THREONINE PROTEIN KINASE
; PROTEIN KINASE; PURINE
MISCELLANEOUS TERMS: CONSERVED INTRACELLULAR SIGNALLING CASCADE; GENE

REGULATION; HUMAN EMBRYONIC KIDNEY 293 CELLS; MITOGEN-ACTIVATED PROTEIN
KINASE; POLYOMAVIRUS ENHANCER; PURINE-RICH-REPEAT-BINDING PROTEIN
TRANSCRIPTION FACTORS; SERINE-THREONINE PROTEIN KINASE; TRANSCRIPTION
FACTOR NF-KAPPA-B
CONCEPT CODES:
02508 Cytology and Cytochemistry-Human
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10300 Replication, Transcription, Translation
10808 Enzymes-Physiological Studies
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
31500 Genetics of Bacteria and Viruses
33506 Virology-Animal Host Viruses
32600 In Vitro Studies, Cellular and Subcellular
BIOSYSTEMATIC CODES:
02616 Papovaviridae (1993-)
02623 Retroviridae (1993-)
86215 Hominidae

4/9/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08830180 BIOSIS NO.: 199395119531
Identity of GABP with NRF-2, a multisubunit activator of cytochrome oxidase expression, reveals a cellular role for an ETS domain activator of viral promoters.
AUTHOR: Virbasius Joseph V; Virbasius Ching-Man A; Scarpulla Richard C(a)
AUTHOR ADDRESS: (a)Dep. Cell, Molecular Structural Biol., Northwestern Univ. Med. Sch., Chicago, IL 60611**USA
JOURNAL: Genes & Development 7 (3):p380-392 1993
ISSN: 0890-9369
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The ETS domain proteins are a diverse family of transcriptional activators that have been implicated recently in the expression of a number of cell-specific and viral promoters. Nuclear respiratory factor 2 (NRF-2) is a nuclear transcription factor that activates the proximal promoter of the rat cytochrome c oxidase subunit IV (RCO4) gene through tandem sequence elements. These elements conform to the consensus for

high-affinity ETS domain recognition sites. We have now purified NRF-2 to homogeneity from HeLa cells and find that it consists of five polypeptides, only one of which has intrinsic DNA-binding ability. The others participate in the formation of heteromeric complexes with distinct binding properties. NRF-2 also specifically recognizes multiple binding sites in the mouse cychrome c oxidase subunit Vb (MCO5b) gene. As in the functionally related RCO4 gene, tandemly arranged NRF-2 sites are essential for the activity of the proximal MCO5b promoter, further substantiating a role for NRF-2 in respiratory chain expression. Determination of peptide sequences from the various subunits of HeLa NRF-2 reveals a high degree of sequence identity with mouse GA-binding protein (GABP), a multisubunit ETS domain activator of herpes simplex virus immediate early genes. A cellular role in the activation of nuclear genes specifying mitochondrial respiratory function is thus assigned to an ETS domain activator of viral promoters.

REGISTRY NUMBERS: 9001-16-5: CYTOCHROME OXIDASE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Genetics; Metabolism; Microbiology
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Muridae (Muridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
CHEMICALS & BIOCHEMICALS: CYTOCHROME OXIDASE
MISCELLANEOUS TERMS: GA-BINDING PROTEIN; NUCLEAR RESPIRATORY FACTOR; OXIDATIVE PHOSPHORYLATION
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10065 Biochemical Studies-Porphyrins and Bile Pigments
10802 Enzymes-General and Comparative Studies; Coenzymes
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
31500 Genetics of Bacteria and Viruses
33506 Virology-Animal Host Viruses
BIOSYSTEMATIC CODES:
86375 Muridae

4/9/14 (Item 1 from file: 73)

06683631 EMBASE No: 1996348547
Effect of glucagon on the xylitol-induced increase in the plasma concentration and urinary excretion of purine bases
Yamamoto T.; Moriwaki Y.; Takahashi S.; Ohata H.; Yamakita J.-i.; Higashino K.
Third Dept. of Internal Medicine, Hyogo College of Medicine, Mukogawa-cho 1-1,Nishinomiya, Hyogo 663 Japan
Metabolism: Clinical and Experimental (METAB. CLIN. EXP.) (United States) 1996, 45/11 (1354-1359)
CODEN: METAA ISSN: 0026-0495
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

To investigate whether glucagon affects the xylitol-induced increase in the production of purine bases (hypoxanthine, xanthine, and uric acid), the present study was performed with five healthy subjects. Intravenous administration of 300 mL 10% xylitol increased the plasma concentration and urinary excretion of purine bases, erythrocyte concentrations of adenosine monophosphate (AMP) and adenosine diphosphate (ADP), and blood concentrations of glyceraldehyde-3-phosphate (GA3P) + dihydroxyacetone phosphate (DHAP), fructose-1,6-bisphosphate (FBP), and lactic acid; it decreased the blood concentration of pyruvic acid and the plasma concentration and urinary excretion of inorganic phosphate. However, intravenous administration of 1 mg glucagon together with xylitol reduced the xylitol-induced changes in oxypurines, pyruvic acid, GABP + DHAP, and FBP, whereas it promoted the xylitol-induced increase in the urinary excretion of total purine bases and did not affect the xylitol-induced increase in the plasma concentration of total purine bases. In addition, in vitro study demonstrated that sodium pyruvate prevented the xylitol-induced degradation of adenine nucleotides in erythrocytes. These results suggested that gluconeogenesis due to glucagon increased the production of pyruvic acid, accelerated the conversion of NADH to NAD, and thereby prevented both the xylitol-induced degradation of adenine nucleotides in organs similar to erythrocytes and the inhibition of xanthine dehydrogenase in the liver and small intestine, resulting in decreases in the plasma concentration and urinary excretion of oxypurines. However, it was also suggested that in the liver storing glycogen, glucagon-induced glycogenolysis accumulated sugar phosphates, resulting in purine degradation, since the xylitol-induced increase in the NADH/NAD ratio partially blocked glycolysis at the level of GABP dehydrogenase. Therefore, administration of glucagon together with xylitol may synergistically increase purine degradation more than xylitol alone, despite decreases in the plasma concentration and urinary excretion of oxypurines.

DRUG DESCRIPTORS:
*glucagon--pharmacology--pd; *purine--drug concentration--cr; *xylitol
--drug administration--ad; *xylitol--drug dose--do
dihydroxyacetone phosphate--drug concentration--cr; glyceraldehyde 3
phosphate--drug concentration--cr; hypoxanthine--drug concentration--cr;
uric acid--drug concentration--cr; xanthine--drug concentration--cr
MEDICAL DESCRIPTORS:
*purine metabolism; *urinary excretion
adult; article; clinical article; drug blood level; drug effect;
erythrocyte level; gluconeogenesis; glycogenolysis; glycolysis; human; male
; nucleotide metabolism; priority journal; protein degradation; protein
phosphorylation
CAS REGISTRY NO.: 11140-85-5, 62340-29-8, 9007-92-5 (glucagon); 120-73-0 (
purine); 87-99-0 (xylitol); 57-04-5 (dihydroxyacetone phosphate);
142-10-9 (glyceraldehyde 3 phosphate); 68-94-0 (hypoxanthine); 69-93-2
(uric acid); 69-89-6 (xanthine)
SECTION HEADINGS:
029 Clinical and Experimental Biochemistry
037 Drug Literature Index

4/9/15 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09529846 97335984 PMID: 9192779
GABP cooperates with c-Myb and C/EBP to activate the neutrophil
elastase promoter.
Nuchprayoon I; Simkevich CP; Luo M; Friedman AD; Rosmarin AG
Division of Pediatric Oncology, The Johns Hopkins Oncology Center, Johns
Hopkins University, Baltimore, MD, USA.
Blood (UNITED STATES) Jun 15 1997, 89 (12) p4546-54, ISSN 0006-4971
Journal Code: A8G
Contract/Grant No.: R01 HL51388, HL, NHLBI; R29DK44728, DK, NIDDK
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Subfile: AIM; INDEX MEDICUS
Neutrophil elastase (NE) is a serine protease that is transcriptionally
regulated during early myeloid differentiation. The murine NE (mNE)
promoter contains functionally important c-Myb, C/EBP, and ets binding
sites. Deletion of the ets site reduced promoter activity by 90%. Although
the ets transcription factor, PU.1, bound to this ets site, it only
modestly activated the mNE promoter. Here, we show that a second
transcription factor from myeloid cells- GABP -binds to the mNE ets site
but strongly activates the mNE promoter. GABP is a heteromeric

transcription factor complex that consists of GABP alpha, an ets factor, and GABP beta, a Notch-related protein. GABP alpha bound to the mNE ets site and, in turn, recruited GABP beta to form a transcriptionally active complex. GABP alpha and PU.1 competed with each other for binding to the mNE ets site. GABP increased the activity of the mNE promoter sevenfold in U937 myeloid cells. GABP cooperated with c-Myb and C/EBP alpha to activate the mNE promoter more than 85-fold in otherwise nonpermissive, nonhematopoietic NIH 3T3 cells. Thus, GABP binds to the crucial mNE promoter ets site and powerfully activates its expression alone and in cooperation with the transcription factors c-Myb and C/EBP.

Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *DNA-Binding Proteins--physiology--PH; *Leukocyte Elastase --genetics--GE; *Nuclear Proteins--physiology--PH; *Promoter Regions (Genetics); *Proto-Oncogene Proteins--physiology--PH; *Trans-Activators --physiology--PH; *Transcription Factors--physiology--PH; *Transcription, Genetic; Base Sequence; CCAAT-Enhancer-Binding Proteins; DNA-Binding Proteins--chemistry--CH; DNA-Binding Proteins--classification--CL; DNA-Binding Proteins--genetics--GE; Dimerization; Enzyme Induction; Leukocyte Elastase--biosynthesis--BI; Lymphoma, Large-Cell, Diffuse --pathology--PA; Mice; Molecular Sequence Data; Multigene Family; Neoplasm Proteins--biosynthesis--BI; Neoplasm Proteins--chemistry--CH; Neoplasm Proteins--genetics--GE; Phosphorylation ; Protein Binding; Protein Processing, Post-Translational; Protein-Serine-Threonine Kinases --metabolism--ME; Proto-Oncogene Proteins c-myb; Regulatory Sequences, Nucleic Acid; Signal Transduction; Transcription Factors--chemistry--CH; Transcription Factors--classification--CL; Transcription Factors --genetics--GE; Tumor Cells, Cultured

CAS Registry No.: 0 (CCAAT-Enhancer-Binding Proteins); 0 (DNA-Binding Proteins); 0 (GABP binding protein); 0 (Neoplasm Proteins); 0 (Nuclear Proteins); 0 (Proto-Oncogene Proteins); 0 (Proto-Oncogene Proteins c-myb); 0 (Trans-Activators); 0 (Transcription Factors)

Enzyme No.: EC 2.7.10 (Protein-Serine-Threonine Kinases); EC 2.7.10.- (casein kinase II); EC 3.4.21.37 (Leukocyte Elastase)

?s s1 and kinase

552 S1

729466 KINASE

S5 60 S1 AND KINASE

?rd

...examined 50 records (50)

...completed examining records

S6 30 RD (unique items)

?s s6 not py=>2001

30 S6

2913426 PY=>2001

S7 25 S6 NOT PY=>2001

7/9/1 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

11840409 BIOSIS NO.: 199900086518
An enhancer located between the neutrophil elastase and proteinase 3 promoters is activated by Sp1 and an Ets factor.
AUTHOR: Nuchprayoon Issarang; Shang Jing; Simkevich Carl P; Luo Menglin; Rosmarin Alan G; Friedman Alan D(a)
AUTHOR ADDRESS: (a)Johns Hopkins Oncol. Cent., Room 3-109, 600 North Wolfe St., Baltimore, MD 21287**USA
JOURNAL: Journal of Biological Chemistry 274 (2):p1085-1091 Jan. 8, 1999
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The adjacent neutrophil elastase, proteinase 3, and azurocidin genes encode serine proteases expressed specifically in immature myeloid cells. Subclones of a 17-kilobase (kb) murine neutrophil elastase genomic clone were assessed for their ability to stimulate the neutrophil elastase promoter in 32D c13 myeloid cells. Region -9.3 to -7.3 kb stimulated transcription 7-fold, whereas other genomic segments were inactive. This enhancer is located in the second intron of the proteinase-3 gene and so may regulate more than one gene in the myeloid protease cluster. Deletional analysis of the enhancer identified several segments which activated the neutrophil elastase and thymidine kinase promoters 3-6-fold. The most active segment was a 220-base pair region centered at -8.6 kb, which activated transcription 31-fold. This segment contains an Sp1 consensus site, which bound Sp1, flanked by two Ets family consensus sequences, which bound PU.1, GABP , and an Ets factor present in myeloid cell extracts. Mutation of the Sp1-binding site reduced enhancer activity 8-fold in 32D cl3 cells, and mutation of either or both Ets-binding sites reduced activity 3-4-fold. Sp1 activated the distal enhancer 5-fold, GABP 3-fold, and the combination 8-fold in Schneider cells.

REGISTRY NUMBERS: 9004-06-2: ELASTASE; 9001-92-7: PROTEINASE; 9002-06-6:
THYMIDINE KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Enzymology (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Mammalia--Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Schneider cell line (Mammalia); 32D c13 cell line (Muridae)--murine myeloid cells

BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Mammals;
Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: neutrophil elastase; proteinase 3; thymidine kinase ; Ets factor; Sp1; mouse proteinase-3 gene (Muridae
CONCEPT CODES:
10806 Enzymes-Chemical and Physical
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
BIOSYSTEMATIC CODES:
85700 Mammalia-Unspecified
86375 Muridae

7/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11725871 BIOSIS NO.: 199800507602
Structure of human cyclin-dependent kinase inhibitor p19INK4d: Comparison to known ankyrin-repeat-containing structures and implications for the dysfunction of tumor suppressor p16INK4a.
AUTHOR: Baumgartner Roland; Fernandez-Catalan Carlos; Winoto Astar; Huber Robert; Engh Richard A(a); Holak Tad A
AUTHOR ADDRESS: (a)Max Planck Inst. Biochem., D-82152 Martinsried**Germany
JOURNAL: Structure (London) 6 (10):p1279-1290 Oct. 15, 1998
ISSN: 0969-2126
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: The four members of the INK4 gene family (p16INK4a, p15INK4b, p18INK4c and p19INK4d) inhibit the closely related cyclin-dependent kinases CDK4 and CDK6 as part of the regulation of the G1fwdarwS transition in the cell-division cycle. Loss of INK4 gene product function, particularly that of p16INK4a, is found in 10-60% of human tumors, suggesting that broadly applicable anticancer therapies might be based on restoration of p16INK4a CDK inhibitory function. Although much less frequent, defects of p19INK4d have also been associated with human cancer (osteosarcomas). The protein structures of some INK4 family members, determined by nuclear magnetic resonance (NMR) spectroscopy and X-ray techniques, have begun to clarify the functional role of p16INK4a and the dysfunction introduced by the mutations

associated with human tumors. Results: The crystal structure of human p19INK4d has been determined at 1.8 ANG resolution using multiple isomorphous replacement methods. The fold of p19INK4d produces an oblong molecule comprising five approximately 32-residue ankyrin-like repeats. The architecture of the protein demonstrates the high structural similarity within the INK4 family. Comparisons to other ankyrin-repeat-containing proteins (GABPbeta, 53BP2 and myotrophin) show similar structures with comparable hydrogen-bonding patterns and hydrophobic interactions. Such comparisons highlight the splayed beta-loop geometry that is specific to INK4 inhibitors. This geometry is the result of a modified ankyrin structure in the second repeat. Conclusions: Among the INK4 inhibitors, the highest amino acid sequence conservation is found in the helical stacks; this conservation creates a conserved beta-loop geometry specific to INK4 inhibitors. Therefore, in addition to models which predict that the conserved helix alpha6 is responsible for CDK inhibition, a binding mode whereby the loops of INK4 proteins bind to the CDKs should also be considered. A similar loop-based interaction is seen in the complex formed between the ankyrin-repeat-containing protein GABPbeta and GABPalpha This mode of binding would be consistent with the observation that p16INK4a is sensitive to deleterious mutations found throughout this tumor suppressor protein; these mutations probably destabilize the three-dimensional structure.

REGISTRY NUMBERS: 9031-44-1: KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology; Tumor Biology
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans; Mammals; Primates; Vertebrates
CHEMICALS & BIOCHEMICALS: ankyrin-repeat-containing structures--comparative biochemistry; cyclin-dependent kinase ; myotrophin--comparative biochemistry; p16h--comparative biochemistry; p18-INK4c--comparative biochemistry; p18h--comparative biochemistry; p19 INK4d--conserved amino acid sequence, crystal structure, enzyme inhibitor, loop-based interaction; p19h--comparative biochemistry; GABP -beta--comparative biochemistry; 53BP2--comparative biochemistry
MISCELLANEOUS TERMS: amino acid sequence; cell cycle; mutation sensitivity; tumor suppressor p16-INK4a dysfunction
CONCEPT CODES:
02508 Cytology and Cytochemistry-Human
02502 Cytology and Cytochemistry-General

03502 Genetics and Cytogenetics-General
03508 Genetics and Cytogenetics-Human
10060 Biochemical Studies-General
10502 Biophysics-General Biophysical Studies
10802 Enzymes-General and Comparative Studies; Coenzymes
24002 Neoplasms and Neoplastic Agents-General
BIOSYSTEMATIC CODES:
86215 Hominidae

7/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11537322 BIOSIS NO.: 199800318654
Implication of a multisubunit Ets-related transcription factor in synaptic expression of the nicotinic acetylcholine receptor.
AUTHOR: Schaeffer Laurent; Duclert Nathalie; Huchet-Dymanus Monique; Changeux Jean-Pierre(a)
AUTHOR ADDRESS: (a)CNRS UA D1284 'Neurobiologie Moleculaire', Inst. Pasteur, 25 rue du Dr Roux, F-75724 Paris Cedex**France
JOURNAL: EMBO (European Molecular Biology Organization) Journal 17 (11):p 3078-3090 June 1, 1998
ISSN: 0261-4189
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In adult muscle, transcription of the nicotinic acetylcholine receptor (AChR) is restricted to the nuclei located at the neuromuscular junction. The N-box, a new promoter element, was identified recently and shown to contribute to this compartmentalized synaptic expression of the AChR delta- and epsilon-subunits. We demonstrate that the N-box mediates transcriptional activation in cultured myotubes and identify the transcription factor that binds to the N-box as a heterooligomer in myotubes and adult muscle. The GABP (GA-binding protein) alpha-subunit belongs to the Ets family of transcription factors, whereas the beta-subunit shares homology with IkappaB and Drosophila Notch protein. GABP binding specificity to mutated N-box in vitro strictly parallels the sequence requirement for beta-galactosidase targeting to the endplate in vivo. In situ hybridization studies reveal that the mRNAs of both GABP subunits are abundant in mouse diaphragm, with preferential expression of the alpha-subunit at motor endplates. In addition, heregulin increases GABPalpha protein levels and regulates phosphorylation of both subunits in cultured chick myotubes. Finally, dominant-negative mutants of either GABPalpha or GABPbeta block

heregulin-elicited transcriptional activation of the AChR delta and epsilon genes. These findings establish the expected connection with a presynaptic trophic factor whose release contributes to the accumulation of AChR subunit mRNAs at the motor endplate.

REGISTRY NUMBERS: 51-84-3: ACETYLCHOLINE; 191809-35-5D: NEUREGULINS;
9026-43-1: PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Molecular Genetics (Biochemistry and Molecular Biophysics); Nervous System (Neural Coordination)
BIOSYSTEMATIC NAMES: Galliformes--Aves, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: chicken (Galliformes); mouse (Muridae)
ORGANISMS: PARTS ETC: diaphragm--muscular system; motor endplate--nervous system; muscle--muscular system; myotubes--muscular system; synapse
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Birds; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: multisubunit Ets-related transcription factor ; mRNA {messenger RNA}--expression; neuregulins; nicotinic acetylcholine receptor delta-subunit gene--expression; nicotinic acetylcholine receptor epsilon-subunit gene--expression; Drosophila Notch protein; GA-binding protein alpha subunit; I-kappa-B--transcription factor
METHODS & EQUIPMENT: immunoprecipitation--analytical method, methodological approach; in situ hybridization--analytical method, methodological approach; metabolic labeling--analytical method, methodological approach; mitogen-activated protein kinase assay--analytical method, methodological approach; mobility shift assay--analytical method, methodological approach; mobility supershift assay --analytical method, methodological approach; Northern blot--analytical method, methodological approach
MISCELLANEOUS TERMS: synaptic gene expression; transcriptional activation; N-box
CONCEPT CODES:
03506 Genetics and Cytogenetics-Animal
10060 Biochemical Studies-General
17501 Muscle-General; Methods
20501 Nervous System-General; Methods
BIOSYSTEMATIC CODES:
85536 Galliformes
86375 Muridae

7/9/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

11476274 BIOSIS NO.: 199800257606
The GABP -responsive element of the interleukin-2 enhancer is regulated by JNK/SAPK-activating pathways in T lymphocytes.
AUTHOR: Hoffmeyer Angelika; Avots Andris; Flory Egbert; Weber Christoph K; Serfling Edgar; Rapp Ulf R(a)
AUTHOR ADDRESS: (a)Inst. Med. Strahlenkunde Zellforschung, Univ. Wuerzburg, Versbacher Strasse 5, D-97078 Wuerzburg**Germany
JOURNAL: Journal of Biological Chemistry 273 (17):p10112-10119 April 24, 1998
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: T cell activation leads via multiple intracellular signaling pathways to rapid induction of interleukin-2 (IL-2) expression, which can be mimicked by costimulation with 12-O-tetradecanoylphorbol-13-acetate (TPA) and ionomycin. We have identified a distal IL-2 enhancer regulated by the Raf-MEK-ERK signaling pathway, which can be induced by TPA/ionomycin treatment. It contains a dyad symmetry element (DSE) controlled by the Ets-like transcription factor GA-binding protein (GABP), a target of activated ERK. TPA/ionomycin treatment of T cells stimulates both mitogen-activated ERK as well as the stress-activated mitogenactivated protein kinase family members JNK/SAPK and p38. In this study, we investigated the contribution of the stress-activated pathways to the induction of the distal IL-2 enhancer. We show that JNK- but not p38-activating pathways regulate the DSE activity. Furthermore, the JNK/SAPK signaling pathway cooperates with the Raf-MEK-ERK cascade in TPA/ionomycin-induced DSE activity. In T cells, overexpression of SPRK/MLK3 an activator of JNK/SAPK strongly induces DSE-dependent transcription and dominant negative kinases of SEK and SAPK impair TPA/ionomycin-induced DSE activity. Blocking both ERK and JNK/SAPK pathways abolishes the DSE induction. The inducibility of the DSE is strongly dependent on the Ets-core motifs, which are bound by GABP . Both subunits of GABP are phosphorylated upon JNK activation in vivo and three different isoforms of JNK/SAPK, but not p38, in vitro. Our data suggest that GABP is targeted by signaling events from both ERR and JNK/SAPK pathways. GABP therefore is a candidate for signal integration and regulation of IL-2 transcription in T lymphocytes.

REGISTRY NUMBERS: 9031-44-1: KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Immune System (Chemical Coordination and

Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: A3.01 (Hominidae)--human T lymphoma cells; HEK293 (Hominidae) ; NIH-3T3 (Muridae)
ORGANISMS: PARTS ETC: T lymphocyte--activation, blood and lymphatics, immune system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animals; Chordates; Humans;
Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Primates; Rodents; Vertebrates
CHEMICALS & BIOCHEMICALS: interleukin-2 enhancer--GA-binding protein-responsive element regulation, characterization; tpa; DSE--induction; GA-binding protein { GABP }--transcription factor; SPRJ/MLK3--JNK/SAPK activator, overexpression
METHODS & EQUIPMENT: bacterially expressed protein purification--methodological approach, purification method; electrophoretic mobility shift assay--analysis/characterization techniques, electrophoretic techniques, analytical method; immunoblotting--analytical method, methodological approach; immunoprecipitation--precipitation techniques , purification method; kinase assay--analytical method, methodological approach; transient transfection metabolic labeling and reporter gene assays--methodological approach; DNA cloning--methodological approach
MISCELLANEOUS TERMS: intracellular signalling; transcription regulation ; JNK/SAPK-activating pathways
CONCEPT CODES:
34508 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
02506 Cytology and Cytochemistry-Animal
02508 Cytology and Cytochemistry-Human
03506 Genetics and Cytogenetics-Animal
03508 Genetics and Cytogenetics-Human
10300 Replication, Transcription, Translation
10506 Biophysics-Molecular Properties and Macromolecules
10808 Enzymes-Physiological Studies
13012 Metabolism-Proteins, Peptides and Amino Acids
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
15004 Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
17002 Endocrine System-General
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

7/9/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11080683 BIOSIS NO.: 199799701828
Cis-elements required for the demethylation of the mouse M-lysozyme downstream enhancer.
AUTHOR: Schmitz Alexander; Short Marc; Ammerpohl Ole; Asbrand Christian; Nickel Joachim; Renkawitz Rainer(a)
AUTHOR ADDRESS: (a)Genetisches Institut, Justus-Liebig-Universitaet, Heinrich-Buff-Ring 58-62, D35392 Giessen**Germany
JOURNAL: Journal of Biological Chemistry 272 (33):p20850-20856 1997
ISSN: 0021-9258
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The mouse lysozyme downstream enhancer was previously colocalized with the DNase 1-hypersensitive site in the chromatin of mature macrophages. This hypersensitive site was shown to be macrophage differentiation dependent. Demethylation of CpG sequences within the enhancer is correlated with lysozyme expression in mature macrophages. Binding of the GABP heterotetrameric transcription factor to the enhancer core element (MLDE), only seen in vivo on the demethylated MLDE element in macrophages, is inhibited by DNA methylation. Here, we analyzed the DNA sequences required for demethylation. In electrophoretic mobility shift experiments we found that in addition to the complete methylated MLDE the hemimethylated form of the lower strand inhibits GABP binding as well. Therefore, GABP is unlikely to be the mediator of demethylation. In addition, we show by stable DNA transfections of methylated mouse lysozyme enhancer sequences that MLDE-flanking sequences are required for demethylation. We narrowed down these DNA elements to two short regions of 163 and 79 base pairs on either side of the MLDE, each of which is sufficient to mediate demethylation of the GABP site.

REGISTRY NUMBERS: 9002-06-6: THYMIDINE KINASE ; 9040-07-7: CHLORAMPHENICOL
ACETYLTRANSFERASE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Enzymology (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: mouse (Muridae)

BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals;
nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
CHEMICALS & BIOCHEMICALS: THYMIDINE KINASE ; CHLORAMPHENICOL
ACETYLTRANSFERASE
MISCELLANEOUS TERMS: Research Article; BIOCHEMISTRY AND BIOPHYSICS;
BLOOD AND LYMPHATICS; CHLORAMPHENICOL ACETYLTRANSFERASE; CIS-ELEMENT;
DEMETHYLATION; DIFFERENTIATION; DNA; IMMUNE SYSTEM; M-LYSOZYME
DOWNSTREAM ENHANCER; MACROPHAGE; THYMIDINE KINASE ; TRANSFECTION
CONCEPT CODES:
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10506 Biophysics-Molecular Properties and Macromolecules
10806 Enzymes-Chemical and Physical
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
BIOSYSTEMATIC CODES:
86375 Muridae

7/9/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

11069780 BIOSIS NO.: 199799690925
GABP factors bind to a distal interleukin 2 (IL-2) enhancer and contribute to c-Raf-mediated increase in IL-2 induction.
AUTHOR: Avots Andris; Hoffmeyer Angelika; Flory Egbert; Cimanis Alexander; Rapp Ulf R; Serfling Edgar(a)
AUTHOR ADDRESS: (a)Inst. Pathol., Josef-Schneider-Str. 2, D-97080 Wuerzburg **Germany
JOURNAL: Molecular and Cellular Biology 17 (8):p4381-4389 1997
ISSN: 0270-7306
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Triggering of the T-cell receptor-CD3 complex activates two major signal cascades in T lymphocytes, (i) Ca-2+-dependent signal cascades and (ii) protein kinase cascades. Both signal cascades contribute to the induction of the interleukin 2 (IL-2) gene during T-cell activation. Prominent protein kinase cascades are those that activate mitogen-activated protein (MAP) kinases. We show here that c-Raf, which

is at the helm of the classic MAP-Erk cascade, contributes to IL-2 induction through a distal enhancer element spanning the nucleotides from positions -502 to -413 in front of the transcriptional start site of the IL-2 gene. Induction of this distal IL-2 enhancer differs from induction of the proximal IL-2 promoter-enhancer, since it is induced by phorbol esters alone and independent from Ca-2+ signals. In DNA-protein binding studies, we detected the binding of transcription factors GABP -alpha and -beta to a dyad symmetry element (DSE) of the distal enhancer, which is formed by palindromic binding sites of Ets-like factors. Introduction of point mutations suppressing GABP binding to the DSE interfered with the induction of the distal enhancer and the entire IL-2 promoterenhancer, while overexpression of both GABP factors enhanced the IL-2 promoter-enhancer induction. Overexpression of BXB, a constitutive active version of c-Raf, and of further members of the Ras-Raf-Erk signal cascade exerted an increase of GABP -mediated promoter-enhancer induction. In conjunction with previously published data on c-Raf-induced phosphorylation of GABP factors (E. Flory, A. Hoffmeyer, U. Smola, U. R. Rapp, and J. T. Bruder, J. Virol. 70:2260-2268, 1996), these results indicate a contribution of GABP factors to the Raf-mediated enhancement of IL-2 induction during T-cell activation.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Endocrine System (Chemical Coordination and Homeostasis); Genetics
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: JURKAT (Hominidae)--cell line
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; humans; mammals; primates; vertebrates
MISCELLANEOUS TERMS: Research Article; A 3.01 CELL LINE; BINDING; BLOOD AND LYMPHATICS; EXPRESSION; GABP FACTOR; GENE EXPRESSION; HUMAN T LEUKEMIA; HUMAN T LYMPHOMA; IMMUNE SYSTEM; INTERLEUKIN-2; INTERLEUKIN-2 ENHANCER; INTERLEUKIN-2 GENE; MOLECULAR GENETICS; T-CELL; TRANSCRIPTION FACTOR
CONCEPT CODES:
03508 Genetics and Cytogenetics-Human
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
17002 Endocrine System-General
15006 Blood, Blood-Forming Organs and Body Fluids-Blood, Lymphatic and Reticuloendothelial Pathologies
15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and

Reticuloendothelial System
24010 Neoplasms and Neoplastic Agents-Blood and Reticuloendothelial Neoplasms
BIOSYSTEMATIC CODES:
86215 Hominidae

7/9/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10757906 BIOSIS NO.: 199799379051
A domain of TEL conserved in a subset of ETS proteins defines a specific oligomerization interface essential to the mitogenic properties of the TEL-PDGFR-beta oncoprotein.
AUTHOR: Jousset Christine(a); Carron Clemence(a); Boureux Anthony; Quang Christine Tran; Oury Cecile; Dusanter-Fourt Isabelle; Charon Martine; Levin Jonathan; Bernard Olivier; Ghysdael Jacques
AUTHOR ADDRESS: (a)CNRS UMR 146, Institut Curie-Section de Recherche, Centre Universitaire, 91405 Orsay**France
JOURNAL: EMBO (European Molecular Biology Organization) Journal 16 (1):p 69-82 1997
ISSN: 0261-4189
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: TEL is a novel member of the ETS family of transcriptional regulators which is frequently involved in human leukemias as the result of specific chromosomal translocations. We show here by co-immunoprecipitation and GST chromatography analyses that TEL and TEL-derived fusion proteins form homotypic oligomers in vitro and in vivo. Deletion mutagenesis identifies the TEL oligomerization domain as a 65 amino acid region which is conserved in a subset of the ETS proteins including ETS-1, ETS-2, FLI-1, ERG-2 and GABP -alpha in vertebrates and PNTP2, YAN and ELG in Drosophila. TEL-induced oligomerization is shown to be essential for the constitutive activation of the protein kinase activity and mitogenic properties of TEL-platelet derived growth factor receptor beta (PDGFR-beta), a fusion oncoprotein characteristic of the leukemic cells of chronic myelomonocytic leukemia harboring a t(5;12) chromosomal translocation. Swapping experiments in which the TEL oligomerization domain was exchanged by the homologous domains of representative vertebrate ETS proteins including ETS-1, ERG-2 and GABP -alpha show that oligomerization is a specific property of the TEL amino-terminal conserved domain. These results indicate that the amino-terminal domain conserved in a subset of the ETS proteins has evolved to generate a specialized protein-protein interaction interface

which is likely to be an important determinant of their specificity as transcriptional regulators.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Evolution and Adaptation; Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics); Oncology (Human Medicine, Medical Sciences); Physiology
BIOSYSTEMATIC NAMES: Diptera--Insecta, Arthropoda, Invertebrata, Animalia ; Echinoidea--Echinodermata, Invertebrata, Animalia; Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Invertebrata-Unspecified--Invertebrata, Animalia
ORGANISMS: human (Hominidae); invertebrate (Invertebrata - Unspecified); sea urchin (Echinoidea); Diptera (Diptera); Invertebrata (Invertebrata - Unspecified)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; arthropods; chordates ; echinoderms; humans; insects; invertebrates; mammals; primates; vertebrates
MOLECULAR SEQUENCE DATABANK NUMBER: amino acid sequence; molecular sequence data; NCBI-L19541
MISCELLANEOUS TERMS: Research Article; AMINO-TERMINAL CONSERVED DOMAIN; BLOOD AND LYMPHATIC DISEASE; CONSERVED; DROSOPHILA-MELANOGASTER; ETS; ETS PROTEINS; GENETICS; LEUKEMIA; MITOGEN; NEOPLASTIC DISEASE; OLIGOMERIZATION INTERFACE; PROTEIN-PROTEIN INTERACTION; SEQUENCE HOMOLOGY; TEL-PDGFR-BETA ONCOPROTEIN; TEL-PLATELET-DERIVED GROWTH FACTOR-RECEPTOR-BETA ONCOPROTEIN; TRANSCRIPTION REGULATOR; TUMOR BIOLOGY
CONCEPT CODES:
01500 Evolution
03506 Genetics and Cytogenetics-Animal
03508 Genetics and Cytogenetics-Human
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10300 Replication, Transcription, Translation
12003 Physiology, General and Miscellaneous-Comparative (1970-)
24007 Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis
64048 Invertebrata, Comparative and Experimental Morphology, Physiology and Pathology-Echinodermata
64076 Invertebrata, Comparative and Experimental Morphology, Physiology and Pathology-Insecta-Physiology

BIOSYSTEMATIC CODES:
34000 Invertebrata-Unspecified
75314 Diptera
83300 Echinoidea
86215 Hominidae

7/9/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10368811 BIOSIS NO.: 199698823729
GABP mediates insulin-increased prolactin gene transcription.
AUTHOR: Ouyang Liaohan(a); Jacob Kirsten K; Stanley Frederick M
AUTHOR ADDRESS: (a)Mt. Sinai Med. Cent., Asher Levy Place, New York, NY 10029**USA
JOURNAL: Journal of Biological Chemistry 271 (18):p10425-10428 1996
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The insulin-response element from the prolactin gene is identical to the Ets-binding site, and dominant-negative Ets protein inhibits insulin-increased prolactin gene expression. Immunoblotting identified the Ets-related transcription factor GABP in nuclear extracts from GH cells. Expression of GABP -alpha and GABP -beta-1 squelches insulin-increased prolactin gene expression. GABP -alpha and GABP -beta-1 bind the insulin-response element of the prolactin promoter, and anti- GABP -alpha and anti- GABP -beta-1 antibodies supershift a species seen with nuclear extracts from GH cells. GABP -alpha immunoprecipitated from insulin-treated, 32P-labeled GH cells was phosphorylated 3-fold more than GABP -alpha from control cells. There was no increase in phosphorylation of GABP -beta in response to insulin. Mitogen-activated protein (MAP) kinase activity is increased 10-fold in insulin-treated GH4 cells. MAP kinase immunoprecipitated from control cells does not phosphorylate GABP -alpha while MAP kinase immunoprecipitated from insulin-treated cells shows substantial phosphorylation of GABP -alpha. These studies suggest that GABP mediates insulin-increased transcription of the prolactin gene. GABP may be regulated by MAP kinase phosphorylation.

REGISTRY NUMBERS: 9002-62-4: PROLACTIN; 9026-43-1: PROTEIN KINASE
DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics);

Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: Muridae (Muridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; mammals;
nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
CHEMICALS & BIOCHEMICALS: PROLACTIN; PROTEIN KINASE
MISCELLANEOUS TERMS: GENE REGULATION; GH4 CELLS; INSULIN-RESPONSE
ELEMENT; MITOGEN-ACTIVATED PROTEIN KINASE ; PROMOTER; SIGNAL TRANSDUCTION
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03506 Genetics and Cytogenetics-Animal
10300 Replication, Transcription, Translation
10808 Enzymes-Physiological Studies
17008 Endocrine System-Pancreas
17014 Endocrine System-Pituitary
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
BIOSYSTEMATIC CODES:
86375 Muridae

7/9/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10320360 BIOSIS NO.: 199698775278
Raf-1 kinase targets GA-binding protein in transcriptional regulation of the human immunodeficiency virus type 1 promoter.
AUTHOR: Flory Egbert; Hoffmeyer Angelika; Smola Ute; Rapp Ulf R(a); Bruder Joseph T
AUTHOR ADDRESS: (a)Inst. Radiobiol. Cell Res., Univ. Wuerzburg, D-97078 Wuerzburg**Germany
JOURNAL: Journal of Virology 70 (4):p2260-2268 1996
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The serine/threonine protein kinase Raf-1 is a component of a conserved intracellular signaling cascade that controls responses to various extracellular stimuli. Transcription from several promoters, including the oncogene-responsive element in the polyomavirus enhancer,

the c-fos promoter, as well as other AP-1- and Ets-dependent promoters, can be induced by Raf-1 kinase . Previously, we have shown that activated Raf-I kinase transactivates the human immunodeficiency virus type 1 (HIV-1) long terminal repeat and have identified the NF-kappa-B binding motif as a Raf-1-responsive element (RafRE). We now report that Raf-1 kinaseinduced transactivation from the HIV RafRE involves the purine-rich-repeat-binding protein (GABP), which is composed of two distinct subunits (alpha- and beta). GABP alpha is an Ets oncogene-related DNA-binding protein, and GABP beta contains four ankyrin-like repeats that have been shown to be essential in protein-protein interactions. In electrophoretic mobility shift assays using nuclear extracts from human Jurkat T cells, a protein-DNA complex which was supershifted with antiserum against GABP alpha and GABP beta was observed. Purified recombinant GABP alpha and beta interact with the HIV RafRE as judged from DNA binding assays. Cotransfection experiments with GABP alpha and beta and Raf-1 kinase demonstrate synergistic transactivation of the HIV-1 promoter. Point mutations in the HIV RafRE abolished the Raf-1 kinase - as well as GABP alpha- and beta-induced transactivation. The observed Raf-1- GABP synergism presumably involves phosphorylation of GABP subunits, phosphorylation of GABP in vivo. However, GABP is not a target of Raf-1 kinase ; instead, it is a substrate of mitogen-activated protein kinase (MAPK/ERK), since in vitro phosphorylation of GABP alpha and beta was achieved by the reconstituted protein kinase cascade but not with purified Raf-1 or MEK. These results suggest that Raf-1 kinase -induced activation of the HIV-1 promoter is mediated by the classical cytoplasmic cascade resulting in MAPK/ERK-mediated phosphorylation of GABP alpha and beta. Because the HIV RafRE corresponds to a region within the promoter which is essential for regulation of HIV-1 expression, the data indicate that in addition to NF-kappa-B, GABP transcription factors are important for induced expression of HIV.

REGISTRY NUMBERS: 139691-76-2: RAF-1 KINASE ; 9026-43-1: SERINE-THREONINE
PROTEIN KINASE ; 9026-43-1: PROTEIN KINASE ; 120-73-0: PURINE
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Cell Biology; Enzymology (Biochemistry and Molecular Biophysics); Genetics; Metabolism; Microbiology; Molecular Genetics (Biochemistry and Molecular Biophysics)
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Papovaviridae--Viruses; Retroviridae--Viruses
ORGANISMS: Hominidae (Hominidae); Papovaviridae (Papovaviridae); Retroviridae (Retroviridae)
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): animals; chordates; humans; mammals; microorganisms; primates; vertebrates; viruses

CHEMICALS & BIOCHEMICALS: RAF-1 KINASE ; SERINE-THREONINE PROTEIN
KINASE ; PROTEIN KINASE ; PURINE
MISCELLANEOUS TERMS: CONSERVED INTRACELLULAR SIGNALLING CASCADE; GENE
REGULATION; HUMAN EMBRYONIC KIDNEY 293 CELLS; MITOGEN-ACTIVATED PROTEIN
KINASE ; POLYOMAVIRUS ENHANCER; PURINE-RICH-REPEAT-BINDING PROTEIN
TRANSCRIPTION FACTORS; SERINE-THREONINE PROTEIN KINASE ; TRANSCRIPTION
FACTOR NF-KAPPA-B
CONCEPT CODES:
02508 Cytology and Cytochemistry-Human
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
10300 Replication, Transcription, Translation
10808 Enzymes-Physiological Studies
13014 Metabolism-Nucleic Acids, Purines and Pyrimidines
31500 Genetics of Bacteria and Viruses
33506 Virology-Animal Host Viruses
32600 In Vitro Studies, Cellular and Subcellular
BIOSYSTEMATIC CODES:
02616 Papovaviridae (1993-)
02623 Retroviridae (1993-)
86215 Hominidae

7/9/10 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09347844 Genuine Article#: 395RL Number of References: 56
Title: Sequence and functional properties of Ets genes in the model organism Drosophila
Author(s): Hsu T (REPRINT) ; Schulz RA
Corporate Source: Med Univ S Carolina,Ctr Mol & Struct Biol, Hollings Canc Ctr,86 Jonathan Lucas St/Charleston//SC/29425 (REPRINT); Med Univ S Carolina,Ctr Mol & Struct Biol, Hollings Canc Ctr,Charleston//SC/29425; Med Univ S Carolina,Dept Cell Biol & Anat,Charleston//SC/29425; Univ Texas,MD Anderson Canc Ctr, Dept Biochem & Mol Biol, Program Genes & Dev,Houston//TX/77030
Journal: ONCOGENE, 2000, V19, N55 (DEC 18), P6409-6416
ISSN: 0950-9232 Publication date: 20001218
Publisher: NATURE PUBLISHING GROUP, HOUNDMILLS, BASINGSTOKE RG21 6XS,

HAMPSHIRE, ENGLAND
Language: English Document Type: ARTICLE
Geographic Location: USA
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; ONCOLOGY; CELL
BIOLOGY; GENETICS & HEREDITY
Abstract: Detailed molecular and genetic studies, coupled with the recent sequencing of the fly genome, have identified eight Ets-related genes in the model organism Drosophila. All show homology to genes in vertebrate species. Functional analyses of some of the Drosophila ets genes have revealed their essential roles in developmental processes such as metamorphosis, oogenesis, neurogenesis, myogenesis, and eye development. Such studies have yielded important insights into our understanding of the genetic control of hormonally-regulated gene expression, programmed cell death, and signal transduction during cell fate determination and differentiation. The developmental roles of E74 (ELF1), pointed (Ets 1), yan (TEL), and D-elg (GABP alpha) will be reviewed in this article, The context of their participation in signal transduction and gene regulation will also be discussed. The information should be of significant value to the study of related processes in higher organisms due to the growing evidence for the cross species conservation of developmental mechanisms.
Descriptors--Author Keywords: Drosophila ; ets ; pointed ; yan ; D-elg ; E74
Identifiers--KeyWord Plus(R): DOMAIN TRANSCRIPTION FACTOR; GROWTH-FACTOR RECEPTOR; EGF RECEPTOR; VENTRAL NEUROBLASTS; MAP KINASE ; FACTOR CF2; D-ELG; OOGENESIS; PATHWAY; ACTIVATION
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DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08910437 Genuine Article#: 342ZC Number of References: 66
Title: The ets transcription factor GABP is required for postsynaptic differentiation in vivo
Author(s): Briguet A; Ruegg MA (REPRINT)
Corporate Source: UNIV BASEL,BIOCTR, DEPT PHARMACOL NEUROBIOL, KLINGELBERGSTR 70/CH-4056 BASEL//SWITZERLAND/ (REPRINT); UNIV BASEL,BIOCTR, DEPT PHARMACOL NEUROBIOL/CH-4056 BASEL//SWITZERLAND/
Journal: JOURNAL OF NEUROSCIENCE, 2000, V20, N16 (AUG 15), P5989-5996
ISSN: 0270-6474 Publication date: 20000815
Publisher: SOC NEUROSCIENCE, 11 DUPONT CIRCLE, NW, STE 500, WASHINGTON, DC 20036
Language: English Document Type: ARTICLE
Geographic Location: SWITZERLAND
Subfile: CC LIFE--Current Contents, Life Sciences;
Journal Subject Category: NEUROSCIENCES
Abstract: At chemical synapses, neurotransmitter receptors are concentrated in the postsynaptic membrane. During the development of the neuromuscular junction, motor neurons induce aggregation of acetylcholine receptors (AChRs) underneath the nerve terminal by the redistribution of existing AChRs and preferential transcription of the AChR subunit genes in subsynaptic myonuclei. Neural agrin, when expressed in nonsynaptic regions of muscle fibers in vivo, activates both mechanisms resulting in the assembly of a fully functional postsynaptic apparatus. Several lines of evidence indicate that synaptic transcription of AChR genes is primarily dependent on a promoter element called N-box. The Ets-related transcription factor growth-associated binding protein (GABP) binds to this motif and has thus been suggested to regulate synaptic gene expression. Here, we assessed the role of GABP in synaptic gene expression and in the formation of postsynaptic specializations in vivo by perturbing its function during postsynaptic differentiation induced by neural agrin. We find that neural agrin-mediated activation of the AChR epsilon subunit promoter is abolished by the inhibition of GABP function. Importantly, the number of AChR aggregates formed in response to neural agrin was strongly reduced. Moreover, aggregates of acetylcholine esterase and utrophin, two additional components of the postsynaptic apparatus, were also reduced. Together, these results are the first direct in vivo evidence that GABP regulates synapse-specific gene expression at the neuromuscular junction and that GABP is required for the formation of a functional postsynaptic apparatus.
Descriptors--Author Keywords: GABP ; transcription ; dominant-negative ; neuromuscular junction ; synapse ; acetylcholine receptor
Identifiers--KeyWord Plus(R): EPSILON-SUBUNIT GENE; VERTEBRATE

NEUROMUSCULAR-JUNCTION; SKELETAL-MUSCLE FIBERS; PROTEIN-KINASE -C;
NICOTINIC ACETYLCHOLINE-RECEPTOR; CONGENITAL MYASTHENIC SYNDROME; N-BOX
MOTIF; BINDING-PROTEIN; SYNAPTIC EXPRESSION; ELECTRICAL-ACTIVITY

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7/9/12 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08271544 Genuine Article#: 264NG Number of References: 40
Title: Synergistic transcriptional activation by hGABP and select members of the activation transcription factor/cAMP response element-binding protein family
Author(s): Sawada J; Simizu N; Suzuki F; Sawa C; Goto M; Hasegawa M; Imai T ; Watanabe H; Handa H (REPRINT)
Corporate Source: TOKYO INST TECHNOL,FRONTIER COLLABORAT RES CTR, RES FUNCT
BIOTECHNOL, MIDORI KU, 4259 NAGATSUT/YOKOHAMA/KANAGAWA 2268501/JAPAN/
(REPRINT); TOKYO INST TECHNOL,FRONTIER COLLABORAT RES CTR, RES FUNCT

BIOTECHNOL, MIDORI KU/YOKOHAMA/KANAGAWA 2268501/JAPAN/; TOKYO INST
TECHNOL,GRAD SCH BIOSCI & BIOTECHNOL, DEPT BIOL INFORMAT, MIDORI
KU/YOKOHAMA/KANAGAWA 2268501/JAPAN/
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1999, V274, N50 (DEC 10), P 35475-35482
ISSN: 0021-9258 Publication date: 19991210
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE
PIKE, BETHESDA, MD 20814
Language: English Document Type: ARTICLE
Geographic Location: JAPAN
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: The Ets-related DNA-binding protein human GA-binding protein (hGABP) alpha interacts with the four ankyrin-type repeats of hGABP beta to form an hGABP tetrameric complex that stimulates transcription through the adenovirus early 4 (E4) promoter. Using co-transfection assays, this study demonstrated that the hGABP complex mediated efficient activation of transcription from E4 promoter synergistically with activating transcription factor (ATF) 1 or cAMP response element-binding protein (CREB), but not ATF2/CRE-BP1. This synergy also partially occurred when hGABP alpha was used alone in place of the combination of hGABP alpha and hGABP beta. hGABP activated an artificial promoter containing only ATF/CREB-binding sites under coexistence of ATF1 or CREB. Consistent with these results, physical interactions of hGABP alpha with ATF1 or CREB were observed in vitro. Functional domain analyses of the physical interactions revealed that the amino-terminal region of hGABP alpha bound to the DNA-binding domain of ATF1, which resulted in the formation of ternary complexes composed of ATF1, hGABP alpha, and hGABP beta. In contrast to hGABP alpha, hGABP beta did not significantly interact with ATF1 and CREB. Taken together, these results indicate that hGABP functionally interacts with selective members of the ATF/CREB family, and also suggest that synergy results from multiple interactions which mediate stabilization of large complexes within the regulatory elements of the promoter region, including DNA-binding and non-DNA-binding factors.
Identifiers--KeyWord Plus(R): FACTOR E4TF1; HUMAN-CHROMOSOME; 2 SUBUNITS;
FACTOR ATF; KINASE -II; RB GENE; ETS; COMPLEX; IDENTIFICATION; EXPRESSION
Cited References:
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7/9/13 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08238086 Genuine Article#: 260XW Number of References: 61
Title: Transcriptional regulation of fas gene expression by GA-binding

protein and AP-1 in T cell antigen receptor CD3 complex-stimulated T cells

Author(s): Li XR; Chong ASF; Wu JM; Roebuck KA; Kumar A; Parrillo JE; Rapp UR; Kimberly RP; Williams JW; Xu XL (REPRINT)

Corporate Source: RUSH PRESBYTERIAN ST LUKES MED CTR,DEPT GEN SURG, 1653 W CONGRESS PKWY/CHICAGO//IL/60612 (REPRINT); RUSH PRESBYTERIAN ST LUKES MED CTR,DEPT GEN SURG/CHICAGO//IL/60612; RUSH PRESBYTERIAN ST LUKES MED CTR,DEPT IMMUNOL MICROBIOL/CHICAGO//IL/60612; RUSH PRESBYTERIAN ST LUKES MED CTR,DEPT MED/CHICAGO//IL/60612; UNIV ALABAMA,DEPT MED, SCH MED, DIV CLIN IMMUNOL & RHEUMATOL/BIRMINGHAM//AL/35284; UNIV WURZBURG,INST MED RADIAT RES & CELL BIOL/D-97080 WURZBURG//GERMANY/

Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1999, V274, N49 (DEC 3), P 35203-35210

ISSN: 0021-9258 Publication date: 19991203

Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814

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Geographic Location: USA; GERMANY

Subfile: CC LIFE--Current Contents, Life Sciences

Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY

Abstract: Fas (CD95 or APO-1), a transmembrane cell surface receptor of the tumor necrosis factor receptor family, is up-regulated in activated T lymphocytes. Our present study identified an upstream enhancer element (between nucleotide positions -862 and -682) containing a GA-binding protein (GABP) site and a low affinity activating protein-1 (AP-l)-binding site. T cell activation increased the DNA binding of GABP and AP-1 to this enhancer site. The specificity of GABP and AP-1 binding was demonstrated by competition electrophoretic mobility shift assay and supershift electrophoretic mobility shift assay with antibodies against GABP and AP-1, respectively. Mutational analysis of Fas promoter revealed that both GABP - and AP-l-binding sites were required for initiating Fas gene transcription. We further show that anti-CD3 mAb, phorbol 12-myristate 13-acetate, and phorbol l2-myristate 13-acetate/ionomycin strongly activated promoters carrying multiple copies of the Fas enhancer, and mutation of either the GABP or AP-1 binding site severely reduced transcriptional activity. Taken together, these results suggest that the transcription factors GABP and AP-1 play a critical role in the induction of Fas gene expression in T cell antigen receptor CD3-stimulated Jurkat cells.

Identifiers--KeyWord Plus(R): SIGNAL-TRANSDUCTION PATHWAY; LIGAND EXPRESSION; MAP KINASE ; INDUCED APOPTOSIS; RESPONSE ELEMENT; CYCLOSPORINE-A; DEATH FACTOR; ACTIVATION; PROMOTER; CD95

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FARIS M, 1998, V18, P5414, MOL CELL BIOL
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7/9/14 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07609950 Genuine Article#: 187RP Number of References: 54
Title: Identification of a GABP alpha/beta binding site involved in the induction of oxytocin receptor gene expression in human breast cells. Potentiation by c-Fos/c-Jun
Author(s): Hoare S; Copland JA; Wood TG; Jeng YJ; Izban MG; Soloff MS (REPRINT)
Corporate Source: UNIV TEXAS,MED BRANCH, DEPT OBSTET & GYNECOL, 301 UNIV BLVD/GALVESTON//TX/77555 (REPRINT); UNIV TEXAS,MED BRANCH, DEPT OBSTET & GYNECOL/GALVESTON//TX/77555; UNIV TEXAS,MED BRANCH, SEALY CTR MOL SCI/GALVESTON//TX/77555
Journal: ENDOCRINOLOGY, 1999, V140, N5 (MAY), P2268-2279
ISSN: 0013-7227 Publication date: 19990500
Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4110
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences

Journal Subject Category: ENDOCRINOLOGY & METABOLISM

Abstract: Oxytocin (OT) receptors (OTRs) mediate reproductive functions, including the initiation of labor and milk ejection. OTR messenger RNA levels are highly regulated, reaching the greatest concentration in the uterus at the end of gestation, and in the mammary gland during lactation. Factors directly effecting changes in OTR gene expression in the mammary gland are not known, so the present studies were done to elucidate possible regulators by characterizing the human OTR gene promoter and 5'-flanking sequence. By analyzing expression of promoter-luciferase constructs, we localized a region between -85 and -65 that was required for both basal and serum-induced expression in a mammary tumor cell line (Hs578T) that expresses inducible, endogenous OTRs. This DNA region contains an ets family target sequence (5'-GGA-3'), and a CRE/AP-1-like motif. The specific Ets factor binding to the OTR promoter was identified, by electrophoretic mobility immunoshift assays, to be GABP alpha/beta. Cotransfection of a -85 OTR/luciferase construct with vectors expressing GABP alpha and GABP beta 1 had only a modest effect on expression, but cotransfection with GABP alpha/beta- with c-Fos/c-Jun-expressing plasmids resulted in an increase of almost 10-fold in luciferase activity. Mutation of either the GABP - or CRE-like binding sites obliterated the induction. These findings are consistent with the involvement of protein kinase C activity in serum induction of the endogenous gene in Hs578T cells. We showed the requirement for GABP alpha/beta and c-Fos/c-Jun in endogenous OTR gene expression using oligonucleotide GABP and AP-1 binding decoys to inhibit serum-induced increases in I-125-labeled OT antagonist binding to Hs578T cells. Our work is the first characterization of the proximal promoter region of the human OTR gene, and it sets the stage for studying regulation of OTR expression in breast cells.

Identifiers--KeyWord Plus(R): AP-1 TRANSCRIPTION FACTORS; ACTIVATION DOMAIN; DNA-SEQUENCES; ESTROUS-CYCLE; T-CELLS; ETS; PROTEIN; PROMOTER; PARTURITION; SUBUNITS

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7/9/15 (Item 6 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07149236 Genuine Article#: 129KE Number of References: 78
Title: Synapse-specific and neuregulin-induced transcription require an Ets site that binds GABP alpha/ GABP beta
Author(s): Fromm L; Burden SJ (REPRINT)
Corporate Source: NYU,MED CTR, SKIRBALL INST, MOL NEUROBIOL PROGRAM, 550 1ST AVE/NEW YORK//NY/10016 (REPRINT); NYU,MED CTR, SKIRBALL INST, MOL NEUROBIOL PROGRAM/NEW YORK//NY/10016
Journal: GENES & DEVELOPMENT, 1998, V12, N19 (OCT 1), P3074-3083
ISSN: 0890-9369 Publication date: 19981001
Publisher: COLD SPRING HARBOR LAB PRESS, 1 BUNGTOWN RD, PLAINVIEW, NY 11724
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: DEVELOPMENTAL BIOLOGY; GENETICS & HEREDITY
Abstract: Localization of acetylcholine receptors (AChRs) to neuromuscular synapses is mediated by multiple pathways. Agrin, which is the signal for one pathway, stimulates a redistribution of previously unlocalized AChRs to synaptic sites. The signal for a second pathway is not known, but this signal stimulates selective transcription of AChR genes in myofiber nuclei located near the synaptic site. Neuregulin (NRG) is a good candidate for the extracellular signal that induces synapse-specific gene expression, since NRG is concentrated at synaptic sites and activates AChR gene expression in cultured muscle cells. Previous studies have demonstrated that 181 bp of 5' flanking DNA from the AChR delta-subunit gene are sufficient to confer synapse-specific transcription in transgenic mice and NRG responsiveness in cultured muscle cells, but the critical sequences within this cis-acting regulatory region have not been identified. We transfected AChR delta-subunit-hGH gene fusions into a muscle cell line, and we show that a potential binding site for Ets proteins is required for NRG-induced gene expression. Furthermore, we produced transgenic mice carrying AChR delta-subunit-hGH gene fusions with a mutation in this NRG-response element (NRE), and we show that this NRE is necessary for synapse-specific transcription in mice. The NRE binds proteins in myotube nuclear extracts, and nucleotides that are important for NRG responsiveness are likewise critical for formation of the protein-DNA complex. This complex contains GABP alpha, an Ets protein, and GABP beta, a protein that lacks an Ets domain but dimerizes with GABP alpha, because formation of the protein-DNA complex is inhibited by antibodies to either GABP alpha or GABP beta. These results

demonstrate that synapse-specific and NRG-induced gene expression require an Ets-binding site and suggest that GABP alpha/ GABP beta mediates the transcriptional response of the AChR delta-subunit gene to synaptic signals, including NRG.

Descriptors--Author Keywords: Ets proteins ; GABP ; neuregulin ; ErbBs ; acetylcholine receptor ; neuromuscular synapse

Identifiers--KeyWord Plus(R): RECEPTOR EPSILON-SUBUNIT; DUCHENNE MUSCULAR-DYSTROPHY; ADULT MUSCLE-FIBERS; NEUROMUSCULAR-JUNCTION;
GENE-EXPRESSION; MAP KINASE ; ELECTRICAL-ACTIVITY; IN-VIVO; DIFFERENTIAL EXPRESSION; CARDIAC DEVELOPMENT

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7/9/16 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

06315469 Genuine Article#: YJ141 Number of References: 25
Title: Elevated expression of ETS-1 gene in a metastatic, tumorigenic human prostate epithelial cell line transformed by the v-Ki-ras oncogene
Author(s): Chen ZQ; Fisher RJ; Li BQ; Kamata T; Kung HF; Lautenberger JA; Rhim JS (REPRINT)
Corporate Source: NCI,LAB BIOCHEM PHYSIOL, BLDG 567, RM 152/FREDERICK//MD/21702 (REPRINT); NCI,LAB BIOCHEM PHYSIOL/FREDERICK//MD/21702; NCI,LAB GENOM DIVERS/FREDERICK//MD/21702; NCI,FREDERICK CANC RES & DEV CTR, IRSP, SAIC FREDERICK/FREDERICK//MD/21702
Journal: INTERNATIONAL JOURNAL OF ONCOLOGY, 1997, V11, N6 (DEC), P1179-1184
ISSN: 1019-6439 Publication date: 19971200
Publisher: INT JOURNAL ONCOLOGY, C/O PROFESSOR D A SPANDIDOS, EDITORIAL OFFICE, 1, S MERKOURI ST, ATHENS 116 35, GREECE
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: ONCOLOGY
Abstract: A suitable in vitro model system to investigate mechanisms of human prostate carcinogenesis is much needed. We have previously demonstrated that an immortal, but non-tumorigenic, human prostate epithelial cell line (267B(1)) can be malignantly transformed by the v-Ki-ras oncogene, and it can serve as a useful model for investigation of the progression steps of prostate carcinogenesis. In this study, we report for the first time the invasive/metastatic phenotype of the v-Ki-ras transformed 267B, cells (267B(1)/Ki-ras). In addition, comparing non-tumorigenic 267B, and metastatic tumorigenic 267B(1)/Ki-ras human prostate epithelial cell lines, we have found that expression of ETS-1 and ERGB mRNA was elevated to 2-5 fold in the metastatic and tumorigenic 267B(1)/Ki-ras cell line. A specific ETS-1 monoclonal antibody E44 also revealed that the expression of ETS-1 protein level in 267B(1)/Ki-ras cell line was higher than those in 267B, cell line. However, other members of the ETS gene family such as ETS-2, GABP alpha and their mRNA expression levels were similar in both cell lines. The activation of MAP kinase , a downstream target for Ki-ras, was also shown. The expression of urokinase plasminogen activator (u-PA) was also increased in the metastatic 267B(1)/Ki-ras line. An obvious capability of invasion was observed in the 267B(1)/Ki-ras cell line, but not in the 267B(1) line using BioCoat

Matrigel invasion chamber assay system. The present study has provided evidence that the v-Ki-ras oncogene may activate the nuclear target gene, ETS-1 gene, to mediate tumorigenic and metastatic capacity of the v-Ki-ras transformed prostate epithelial cells.
Descriptors--Author Keywords: prostate ; ETS-1 gene ; metastasis ; v-Ki-ras ; epithelial cells
Identifiers--KeyWord Plus(R): TRANSCRIPTION FACTOR; MUTATIONS; C-ETS1; TRANSACTIVATION; ACTIVATION; CARCINOMA; PROTEINS; FAMILY; CANCER; DNA
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7/9/17 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05896979 Genuine Article#: XE974 Number of References: 51
Title: GABP cooperates with c-Myb and C/EBP to activate the neutrophil elastase promoter
Author(s): Nuchprayoon I; Simkevich CP; Luo ML; Friedman AD; Rosmarin AG (REPRINT)

Corporate Source: MIRIAM HOSP,DIV HEMATOL ONCOL, 164 SUMMIT AVE/PROVIDENCE//RI/02906 (REPRINT); MIRIAM HOSP,DIV HEMATOL ONCOL/PROVIDENCE//RI/02906; JOHNS HOPKINS UNIV,SCH MED, JOHNS HOPKINS ONCOL CTR, DIV PEDIAT ONCOL/BALTIMORE//MD/21205; BROWN UNIV,DEPT MED, DIV HEMATOL/PROVIDENCE//RI/02912
Journal: BLOOD, 1997, V89, N12 (JUN 15), P4546-4554
ISSN: 0006-4971 Publication date: 19970615
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences; CC CLIN--Current Contents, Clinical Medicine
Journal Subject Category: HEMATOLOGY
Abstract: Neutrophil elastase (NE) is a serine protease that is transcriptionally regulated during early myeloid differentiation. The murine NE (mNE) promoter contains functionally important c-Myb, C/EBP, and ets binding sites. Deletion of the ets site reduced promoter activity by 90%. Although the ets transcription factor, PU.1, bound to this ets site, it only modestly activated the mNE promoter. Here, we show that a second transcription factor from myeloid cells - GABP - binds to the mNE ets site but strongly activates the mNE promoter. GABP is a heteromeric transcription factor complex that consists of GABP alpha, an ets factor, and GABP beta, a Notch-related protein. GABP alpha bound to the mNE ets site and, in turn, recruited GABP beta to form a transcriptionally active complex. GABP alpha and PU,1 competed with each other for binding to the mNE ets site. GABP increased the activity of the mNE promoter sevenfold in U937 myeloid cells. GABP cooperated with c-Myb and C/EBP alpha to activate the mNE promoter more than 85-fold in otherwise nonpermissive, nonhematopoietic NIH 3T3 cells. Thus, GABP binds to the crucial mNE promoter ets site and powerfully activates its expression alone and in cooperation with the transcription factors c-Myb and C/EBP. (C) 1997 by The American Society of Hematology.
Identifiers--KeyWord Plus(R): STIMULATING FACTOR-RECEPTOR; TRANSCRIPTION FACTOR PU.1; ETS PROTEINS COOPERATE; BINDING-PROTEIN; DNA-BINDING; LEUCINE-ZIPPER; FAMILY MEMBER; SPI-B; EXPRESSION; GENE
Research Fronts: 95-0059 001 (MAP KINASE ACTIVATION; REGULATION OF THE RAS SIGNALING PATHWAY; 12,020-DALTON PROTEIN)
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05431763 Genuine Article#: BG79B Number of References: 21
Title: PURIFICATION AND CHARACTERIZATION OF GENE-SPECIFIC TRANSCRIPTION
FACTORS - C/EBP, GABP , AND IL-4 STAT
Author(s): YEH WC; HOU JZ; MCKNIGHT SL
Corporate Source: JOHNS HOPKINS UNIV,DEPT BIOL/BALTIMORE//MD/21218
Journal: METHODS IN ENZYMOLOGY, 1996, V274, P101-112
ISSN: 0076-6879
Language: ENGLISH Document Type: REVIEW
Geographic Location: USA
Subfile: Science Citation Index; SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; BIOCHEMICAL
RESEARCH METHODS
Identifiers--KeyWords Plus: GEL-ELECTROPHORESIS; POLYACRYLAMIDE GELS; RNA-POLYMERASE; LEUCINE ZIPPER; BINDING; DNA; PROTEINS; ELEMENT;
IDENTIFICATION; ACTIVATION
Research Fronts: 95-0478 002 (TRANSCRIPTIONAL REGULATION;
CCAAT/ENHANCER-BINDING PROTEIN FAMILY; HUMAN C/EBP-ALPHA GENE; LIVER
GROWTH)
95-5896 002 (ACTIVATION OF THE STAT3 ACUTE-PHASE RESPONSE FACTOR
TRANSCRIPTION FACTOR; HUMAN THYMIDINE KINASE GENE PROMOTER;
DNA-BINDING DOMAIN; PROTEIN COMPLEX)
95-0477 001 (CYTOKINE RECEPTOR SIGNALING MECHANISMS; ACTIVATION OF
MULTIPLE PROTEIN-TYROSINE KINASES; STAT TRANSCRIPTION FACTORS; EARLY
RESPONSE GENES)
95-4415 001 (ELECTROSTATIC REPULSIONS IN THE 2-STRANDED ALPHA-HELICAL

COILED-COIL LEUCINE-ZIPPER; BZIP PROTEINS; TRANSCRIPTION FACTORS;
TRIMERIC STRUCTURAL DOMAIN)
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7/9/19 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05255520 Genuine Article#: VL693 Number of References: 61
Title: REDOX REGULATION OF GA-BINDING PROTEIN-ALPHA DNA-BINDING ACTIVITY
Author(s): MARTIN ME; CHINENOV Y; YU M; SCHMIDT TK; YANG XY
Corporate Source: UNIV MISSOURI,DEPT BIOCHEM/COLUMBIA//MO/65212
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1996, V271, N41 (OCT 11), P 25617-25623
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: We have investigated the reduction/oxidation (redox) regulation of the heteromeric transcription factor GA-binding protein (GABP), GABP , also known as nuclear respiratory factor 2, regulates the

expression of nuclear encoded mitochondrial proteins involved in oxidative phosphorylation, including cytochrome c oxidase subunits IV and Vb, as well as the expression of mitochondrial transcription factor 1. GABP is composed of two subunits, the Ets-related GABP -alpha, which mediates specific DNA binding, and GABP -beta, which forms heterodimers and heterotetramers on DNA sequences containing the PEA3/Ets motif ((C/A)GGA(A/T)(G/A)). We demonstrate here that GABP DNA binding activity and GABP -dependent gene expression in 3T3 cells are inhibited by pro oxidant conditions. DNA binding of recombinant GABP -alpha was activated by chemical reduction (dithiothreitol) and by thioredoxin; however, GSSG inhibited GABP DNA binding activity, Treatment of GABP -alpha, but not GABP -beta(1), with sulfhydryl-alkylating agents also inhibited GABP DNA binding activity, Our results suggest that GABP DNA binding activity is redox-regulated in vivo, possibly by thioredoxin-mediated reduction and by GSSG-mediated oxidation of the GABP -alpha subunit. The regulation of GABP (nuclear respiratory factor 2) DNA binding activity by cellular redox changes provides an important link between mitochondrial and nuclear gene expression and the redox state of the cell.

Identifiers--KeyWords Plus: SUBUNIT-IV GENE; NF-KAPPA-B; MITOCHONDRIAL TRANSCRIPTION FACTOR; ETS-RELATED PROTEINS; SECONDARY STRUCTURE; E4TF1
SUBUNITS; MURINE ETS-1; IN-VITRO; ACTIVATION; DOMAIN

Research Fronts: 94-0019 003 (CONSTITUTIVE NF-KAPPA-B ACTIVITY; HUMAN T-CELL LEUKEMIA-VIRUS TYPE-I TAX ACTIVATION; REGULATION OF HIV EXPRESSION; P50 SUBUNIT)
94-0374 002 (MITOGEN-ACTIVATED PROTEIN- KINASE KINASE ACTIVATOR;
SIGNAL-TRANSDUCTION PATHWAY; INHIBITION OF RAF-1)
94-3389 001 (REDUCED ESCHERICHIA-COLI THIOREDOXIN; RECOMBINANT HUMAN
ADULT T-CELL LEUKEMIA-DERIVED FACTOR; EFFECT OF DISULFIDE BRIDGE
FORMATION)
94-4806 001 (GENE ORGANIZATION; LONG-CHAIN FATTY-ACID TRANSPORT;
TRANSCRIPTION FACTOR)

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7/9/20 (Item 11 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03365546 Genuine Article#: PB104 Number of References: 67
Title: MOLECULAR AND GENETIC-CHARACTERIZATION OF GABP -BETA
Author(s): DELABROUSSE FC; BIRKENMEIER EH; KING DS; ROWE LB; MCKNIGHT SL
Corporate Source: TULARIK INC/S SAN FRANCISCO//CA/94080; TULARIK INC/S SAN
FRANCISCO//CA/94080
Journal: GENES & DEVELOPMENT, 1994, V8, N15 (AUG 1), P1853-1865
ISSN: 0890-9369
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: DEVELOPMENTAL BIOLOGY; GENETICS & HEREDITY
Abstract: This report outlines three observations relating to GABP beta, a polypeptide constituent of the heterotetrameric transcription factor GABP . Evidence is presented showing that the mouse genome encodes two highly related GABP beta polypeptides, designated GABP beta 1-1 and GABP beta 2-1. Genomic and cDNA copies of the newly defined Gabpb2 gene were cloned and characterized, providing the conceptually translated amino acid sequence of GABP beta 2-1. The genes encoding these two proteins, as well as GABP alpha, were mapped to three unlinked chromosomal loci. Although physically unlinked, the patterns of expression of the three genes were strikingly concordant. Finally, the molecular basis of GABP beta dimerization was resolved. Carboxy-terminal regions of the two GABP beta polypeptides, which mediate dimerization, bear highly related primary amino acid sequences. Both sequences are free of alpha-helix destabilizing residues and, when displayed on idealized alpha-helical projections, reveal marked amphipathy. Two observations indicate that these regions adopt an alpha-helical conformation and intertwine as coiled-coils. First, the dimer-forming region of GABP beta 2-1 can functionally replace the leucine zipper of a bZIP transcription factor. Second, a synthetic peptide corresponding to this region shows distinctive helical

properties when examined by circular dichroism spectroscopy. finally, evidence is presented showing that GABP beta 1-1 and GABP beta 2-1 can heterodimerize through this carboxy-terminal domain, but neither protein can heterodimerize via the dimer-forming region of the bZIP protein C/EBP beta.

Descriptors--Author Keywords: GABP -BETA ; TRANSCRIPTION COMPLEXES ; GENE EXPRESSION ; DIMERIZATION ; ALPHA-HELICAL ; COILED-COIL

Identifiers--KeyWords Plus: HEAT-SHOCK FACTOR; GCN4 LEUCINE-ZIPPER; DNA-BINDING DOMAIN; DIMERIZATION SPECIFICITY; COILED COILS; MOUSE CHROMOSOME-16; ACTIVATOR PROTEIN; TRANSCRIPTION; C/EBP; ETS

Research Fronts: 92-0375 007 (FOS JUN DNA-BINDING ACTIVITY; TRANSCRIPTIONAL ACTIVATION; MAP KINASE ; AP-1 SITES; DIFFERENTIAL REGULATION; NUCLEAR PROTEINS)

92-4157 003 (LEUCINE ZIPPER; PROTEIN DNA COMPLEX; TRIPLE-STRANDED ALPHA-HELICAL COILED-COIL; PROTON CHANNEL PEPTIDES; MOLECULAR RECOGNITION)

92-4812 002 (PUTATIVE ANAEROBIC COPROPORPHYRINOGEN-III OXIDASE IN RHODOBACTER-SPHAEROIDES; TRANSCRIPTIONAL REGULATORY ELEMENT; FUNCTIONAL EXPRESSION)

92-1373 001 (SOLID-PHASE PEPTIDE-SYNTHESIS; REAGENT FOR DISULFIDE BOND FORMATION; ARYLSULFONYL SIDE-CHAIN PROTECTED ARGININES)

92-3056 001 (UPTAKE OF SURFACTANT PROTEIN-B; CASEIN KINASE -II; CATALYTIC SUBUNITS)

92-4702 001 (ALPHA-HELIX STABILITY IN PROTEINS; SYNTHETIC MODEL PEPTIDES; CONFORMATIONAL TRANSITION; T4 LYSOZYME; VARYING CHAIN LENGTHS; FOLDING KINETICS)

92-6848 001 (TRANSCRIPTIONAL ACTIVATION DOMAIN; PROMOTER REGION; DNA-BINDING PROTEINS; NUCLEAR FACTORS; RNA POLYMERASE-II)

92-8192 001 (ZINC FINGER PROTEIN; DNA-BINDING ACTIVITY; NF-IL6 TRANSCRIPTION FACTOR; C-FOS SERUM RESPONSE ELEMENT; CDNA CLONING; DIFFERENTIAL EXPRESSION)

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7/9/21 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03221572 Genuine Article#: NP513 Number of References: 72
Title: AN INTRICATE ARRANGEMENT OF BINDING-SITES FOR THE ETS FAMILY OF
TRANSCRIPTION FACTORS REGULATES ACTIVITY OF THE ALPHA-4 INTEGRIN GENE
PROMOTER
Author(s): ROSEN GD; BARKS JL; IADEMARCO MF; FISHER RJ; DEAN DC
Corporate Source: WASHINGTON UNIV,SCH MED,BOX 8052,660 S EUCLID/ST LOUIS//MO/63110; WASHINGTON UNIV,SCH MED/ST LOUIS//MO/63110; WASHINGTON
UNIV,SCH MED,DEPT CELL BIOL/ST LOUIS//MO/63110; PROGRAM RESOURCES
INC,DYNCORP/FREDERICK//MD/21701
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1994, V269, N22 (JUN 3), P 15652-15660
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: alpha 4 integrins mediate cell-cell and cell-extracellular matrix interactions that are critical for maturation and function of the

immune system as well as differentiation of skeletal muscle. Here we examine molecular mechanisms controlling the pattern of alpha 4 expression. The activity of constructs containing 5' deletion mutants of the alpha 4 gene promoter was compared in transfection assays into cell lines that express alpha 4 and cell lines that do not. The sequence between position -42 and -76 base pairs (bp) was required for efficient transcription in cells that express alpha 4, but it showed no activity in HeLa cells, which do not express alpha 4. Three binding sites for the Ets family of transcription factors are found in this region: two adjacent sites at positions -50 and -54 bp and a more 5' site at position -67 bp. Using a series of constructs containing deletions and mutations in this region, we found that the 3'-most site alone was sufficient for binding GA-binding protein alpha (GABP alpha)/ GABP beta and for a low level of transcriptional activation. When all three sites were present, a second complex "a" was detected, which contains an unknown member of the Ets family. Formation of complex a was cell-type specific and correlated with a high level of transcription. Deletion of the 5'-most Ets site had no effect on binding to GABP alpha/ GABP beta, but it eliminated a. Concomitant with this loss of a, a new Ets-1-containing complex "c" appeared. Complex c substituted efficiently for complex a in transcriptional activation. We conclude that although neither of the two 5'-most Ets sites alone binds nuclear protein, they appear to act as modulators which control the pattern of Ets proteins that bind the alpha 4 gene promoter. This arrangement of Ets sites, coupled with the tissue- and developmental-specific expression of Ets members, likely play a key role in defining the pattern of alpha 4 integrin.

Identifiers--KeyWords Plus: STEROID-RECEPTOR SUPERFAMILY; ECDYSONE-INDUCIBLE GENE; LONG TERMINAL REPEAT; DNA-BINDING; C-ETS; LYMPHOID-CELLS; DROSOPHILA-MELANOGASTER; MONOCLONAL-ANTIBODY; ADHESION MOLECULE; LEUKEMIA-VIRUS

Research Fronts: 92-2346 004 (EXPRESSION OF ENDOTHELIAL ADHESION MOLECULES INVIVO; E-SELECTIN LIGAND SPECIFICITIES; CUTANEOUS INFLAMMATION; LYMPHOCYTE INTERACTIONS)

92-7069 002 (ETS GENE FAMILY; DNA-BINDING DOMAIN; DIRECT INTERACTION OF CREB PROTEIN)

92-0022 001 (REL NF-KAPPA-B TRANSCRIPTION FACTORS; DNA-BINDING ACTIVITY; NUCLEAR EXPRESSION; HIV-1 PROMOTER)

92-3377 001 (BETA-1 INTEGRIN RECEPTORS; ADHESION MOLECULES; CELL-SURFACE EXPRESSION)

92-4818 001 (RAT PROLACTIN GENE; RETINOIC ACID IN MCF-7 CELLS; TRANSCRIPTIONAL REGULATION; PROMOTER ACTIVATION; PROTEIN KINASE -A;

FIREFLY LUCIFERASE ACTIVITY)

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7/9/22 (Item 13 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

02061905 Genuine Article#: JY163 Number of References: 51
Title: THE BASAL PROMOTER ELEMENTS OF MURINE CYTOCHROME-C-OXIDASE
SUBUNIT-IV GENE CONSIST OF TANDEMLY DUPLICATED ETS MOTIFS THAT BIND TO
GABP -RELATED TRANSCRIPTION FACTORS
Author(s): CARTER RS; BHAT NK; BASU A; AVADHANI NG
Corporate Source: UNIV PENN,SCH VET MED,DEPT ANIM BIOL,BIOCHEM LABS/PHILADELPHIA//PA/19104; UNIV PENN,SCH VET MED,DEPT ANIM BIOL,BIOCHEM LABS/PHILADELPHIA//PA/19104; NCI,PROGRAM RESOURCES
INC,DYNCORP,FREDERICK CANC RES & DEV CTR,MOLEC ONCOL

LAB/FREDERICK//MD/21702
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1992, V267, N32 (NOV 15), P 23418-23426
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: DNA sequences required for expression of the mouse cytochrome c oxidase subunit IV (COXIV) promoter were identified by transient expression of recombinant COXIV-chloramphenicol acetyltransferase constructs in COS and NIH-3T3 cells. Activity of the COXIV promoter is shown to depend upon upstream Sp1 binding sequences and two tandemly repeated 21-base pair sequence elements each mapping to sites of mRNA initiation. Each initiation region repeat contains a binding site for an ets-related transcription factor which demonstrates specificity for the characteristic GGAA ets sequence motif and reactivity with an ets domain-directed monoclonal pan ets antibody. The two 21-base pair repeats are sufficient for transcriptional activity suggesting that the ets-related factor may be involved in both transcriptional activation and start site positioning. The ets-related protein found in COS nuclear extracts is shown to be identical or closely related to the GA-binding protein (GABP) by comparison of electrophoretic mobilities and immunological reactivities of DNA-protein complexes formed with purified recombinant expressed GABP alpha and beta subunits. Sp1 and the GABP -related factors also bind to another mouse cytochrome oxidase subunit gene COXVb. The similar promoter features of these two genes suggests a possible means of coordinate transcriptional regulation among such respiratory proteins.
Identifiers--KeyWords Plus: UPSTREAM ACTIVATION SITE; RNA POLYMERASE-II;
STRUCTURAL ORGANIZATION; DNA-BINDING; NUCLEOTIDE-SEQUENCE; MITOCHONDRIAL-DNA; NUCLEAR FACTOR; EXPRESSION; PROTEIN; YEAST
Research Fronts: 90-6078 002 (CONSTITUTIVE EXPRESSION OF HIV-1 TAT PROTEIN; DISTAL CIS-ACTING REGULATORY ELEMENTS; CULTURED MOUSE MAMMARY
CELLS; VIRAL ENHANCER; SERUM RESPONSE FACTOR)
90-0429 001 (NF-KAPPA-B TRANSCRIPTION FACTOR; DROSOPHILA HOMEODOMAIN
PROTEINS; C-MYC GENE; EMBRYONIC EXPRESSION PATTERN; POU-SPECIFIC
DOMAIN)
90-1410 001 (MITOCHONDRIAL PROTEIN IMPORT; DEFICIENCY IN 2 YEAST COENZYME-Q MUTANTS; STRUCTURAL GENE ENCODING HEXAPRENYL PYROPHOSPHATE

SYNTHETASE)
90-2447 001 (UPSTREAM PROMOTER ACTIVITY OF THE HUMAN EPSILON-GLOBIN
GENE; HIGH-LEVEL EXPRESSION; TRANSGENIC MICE; RNA POLYMERASE-II; MOUSE
MAMMARY-TUMOR VIRUS-DNA)
90-3495 001 (PURIFIED TRANSCRIPTION FACTORS; MAMMALIAN RNA
POLYMERASE-II; RAT-BRAIN CREATINE- KINASE PROMOTER; FUNCTIONAL
PREINITIATION COMPLEX)
90-6920 001 (MAMMALIAN CYTOCHROME-C-OXIDASE; NUCLEOTIDE-SEQUENCE OF A
MOUSE CDNA; SUBUNIT COMPOSITION; AEROBIC RESPIRATORY-CHAIN;
NUCLEAR-ENCODED GENE)
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7/9/23 (Item 14 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01154044 Genuine Article#: GB295 Number of References: 48
Title: IDENTIFICATION OF ETS-RELATED AND NOTCH-RELATED SUBUNITS IN GA
BINDING-PROTEIN
Author(s): LAMARCO K; THOMPSON CC; BYERS BP; WALTON EM; MCKNIGHT SL
Corporate Source: CARNEGIE INST WASHINGTON,DEPT EMBRYOL,HOWARD HUGHES RES
LABS/BALTIMORE//MD/21210
Journal: SCIENCE, 1991, V253, N5021, P789-792
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC PHYS--Current Contents, Physical, Chemical & Earth Sciences; CC LIFE--Current Contents, Life Sciences; CC AGRI--Current Contents, Agriculture, Biology & Environmental Sciences
Journal Subject Category: MULTIDISCIPLINARY SCIENCES
Abstract: Recombinant cDNA clones that encode two distinct subunits of the transcription factor GA binding protein (GABP) have been isolated. The predicted amino add sequence of one subunit, GABP -alpha, exhibits similarity to the sequence of the product of the ets-1 proto-oncogene

in a region known to encompass the Ets DNA binding domain. The sequence of the second subunit, GABP -beta, contains four 33-amino acid repeats located dose to the NH2-terminus of the subunit. The sequences of these repeats are similar to repeats in several transmembrane proteins, including Notch from Drosophila melanogaster and Glp-1 and Lin-12 from Caenorhabditis elegans. Avid, sequence-specific binding to DNA required the presence of both polypeptides, revealing a conceptual convergence of nuclear transforming proteins and membrane-anchored proteins implicated in developmentally regulated signal transduction processes.

Identifiers--KeyWords Plus: HERPES-SIMPLEX VIRUS; IMMEDIATE-EARLY GENE;
CELL-CYCLE CONTROL; DNA-BINDING; REGULATORY SEQUENCES;
GEL-ELECTROPHORESIS; THYMIDINE KINASE ; INDUCIBLE GENE; GROWTH-FACTOR;
REL ONCOGENE

Research Fronts: 89-1447 002 (DEVELOPMENTALLY REGULATED GENE; CAPPING
PROTEIN; CDNA SEQUENCE; GENOME ORGANIZATION)
89-5548 002 (PROTEIN DNA INTERACTIONS; NUCLEAR FACTORS; UPSTREAM
REGULATORY REGION; MUSCLE-SPECIFIC TRANSCRIPTION; ENHANCER ACTIVITY)
89-4250 001 (TRANSFORMING GROWTH FACTOR-ALPHA; DROSOPHILA PROTEINS;
DIFFERENTIATION OF THE CAENORHABDITIS-ELEGANS TOUCH RECEPTOR NEURONS)

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YOCHEM J, 1988, V335, P547, NATURE

7/9/24 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10192836 99287743 PMID: 10359616
Activation of utrophin promoter by heregulin via the ets-related transcription factor complex GA-binding protein alpha/beta.
Khurana TS; Rosmarin AG; Shang J; Krag TO; Das S; Gammeltoft S
Department of Clinical Biochemistry, University of Copenhagen Medical School, The Glostrup Hospital, Glostrup DK 2600, Denmark. tsk@dcb-glostrup.dk
Molecular biology of the cell (UNITED STATES) Jun 1999, 10 (6) p2075-86, ISSN 1059-1524 Journal Code: BAU
Contract/Grant No.: KO8 NS-01858, NS, NINDS; R29 DK-44728, DK, NIDDK
Languages: ENGLISH
Document type: Journal Article

Record type: Completed
Subfile: INDEX MEDICUS

Utrophin/dystrophin-related protein is the autosomal homologue of the chromosome X-encoded dystrophin protein. In adult skeletal muscle, utrophin is highly enriched at the neuromuscular junction. However, the molecular mechanisms underlying regulation of utrophin gene expression are yet to be defined. Here we demonstrate that the growth factor heregulin increases de novo utrophin transcription in muscle cell cultures. Using mutant reporter constructs of the utrophin promoter, we define the N-box region of the promoter as critical for heregulin-mediated activation. Using this region of the utrophin promoter for DNA affinity purification, immunoblots, in vitro kinase assays, electrophoretic mobility shift assays, and in vitro expression in cultured muscle cells, we demonstrate that ets-related GA-binding protein alpha/beta transcription factors are activators of the utrophin promoter. Taken together, these results suggest that the GA-binding protein alpha/beta complex of transcription factors binds and activates the utrophin promoter in response to heregulin-activated extracellular signal-regulated kinase in muscle cell cultures. These findings suggest methods for achieving utrophin up-regulation in Duchenne's muscular dystrophy as well as mechanisms by which neurite-derived growth factors such as heregulin may influence the regulation of utrophin gene expression and subsequent enrichment at the neuromuscular junction of skeletal muscle.

Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Cytoskeletal Proteins--genetics--GE; *DNA-Binding Proteins --metabolism--ME; *Glycoproteins--metabolism--ME; *Membrane Proteins --genetics--GE; *Nerve Growth Factors--metabolism--ME; *Transcription Factors--metabolism--ME; Base Sequence; Cells, Cultured; Chromatography, Affinity; Cytoskeletal Proteins--metabolism--ME; DNA-Binding Proteins --genetics--GE; Electrophoresis--methods--MT; Gene Expression Regulation; Glycoproteins--pharmacology--PD; Membrane Proteins--metabolism--ME; Mice; Muscle, Skeletal--cytology--CY; Muscle, Skeletal--drug effects--DE; Muscle, Skeletal--metabolism--ME; Nerve Growth Factors--pharmacology--PD; Promoter Regions (Genetics); Rats; Recombinant Proteins--genetics--GE; Recombinant Proteins--metabolism--ME; Signal Transduction; Trans-Activatio n (Genetics); Transcription Factors--genetics--GE

CAS Registry No.: 0 (Cytoskeletal Proteins); 0 (DNA-Binding Proteins) ; 0 (GABP binding protein); 0 (Glycoproteins); 0 (Membrane Proteins); 0 (Nerve Growth Factors); 0 (Recombinant Proteins); 0 (Transcription Factors); 0 (dystrophin-related protein)

Record Date Created: 19990729

7/9/25 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07131643 93135795 PMID: 8422262
Evolution of the ets gene family.
Laudet V; Niel C; Duterque-Coquillaud M; Leprince D; Stehelin D
CNRS UA 1160, Institut Pasteur, Lille, France.
Biochemical and biophysical research communications (UNITED STATES) Jan 15 1993, 190 (1) p8-14, ISSN 0006-291X Journal Code: 9Y8
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Subfile: INDEX MEDICUS
Over the past few years a variety of genes have been described whose protein products share similarity with that of the c-ets-1 proto-oncogene, the cellular counterpart of the v-ets oncogene of the avian E26 retrovirus. This so-called "ets family" of transcription factors includes at least a dozen members present in several organisms. We have questioned the common evolutionary origin of these various gene products. By constructing phylogenetical trees with different methods, we show that the ets family is very ancient since the duplication of the various groups of ets related proteins occurred before the Arthropods/Vertebrates split (ca. 500 million years).
Tags: Animal; Comparative Study; Human
Descriptors: *Evolution; *Multigene Family; *Proto-Oncogene Proteins --genetics--GE; *Proto-Oncogenes; Amino Acid Sequence; Molecular Sequence Data; Phylogeny; Protein-Tyrosine Kinase --genetics--GE; Sequence Homology, Amino Acid
CAS Registry No.: 0 (Proto-Oncogene Proteins); 0 (proto-oncogene protein ets); 0 (proto-oncogene protein ets-2)
Enzyme No.: EC 2.7.1.112 (Protein-Tyrosine Kinase)
Gene Symbol: ist/GeneSymbol D-elg; ist/GeneSymbol D-ets-2; ist/GeneSymbol D-ets-3; ist/GeneSymbol D-ets-4; ist/GeneSymbol D-ets-6; ist/GeneSymbol E74 ; ist/GeneSymbol ELG; ist/GeneSymbol ERG; ist/GeneSymbol ETS; ist/GeneSymbol c-ets-1; ist/GeneSymbol c-ets-2; ist/GeneSymbol elf-1; ist/GeneSymbol elk-1; ist/GeneSymbol erg; ist/GeneSymbol ets; ist/GeneSymbol fli-1; ist/GeneSymbol gabp &agr;; ist/GeneSymbol pea3; ist/GeneSymbol pok; ist/GeneSymbol sap-1; ist/GeneSymbol spi-1; ist/GeneSymbol spiB; ist/GeneSymbol yan
Record Date Created: 19930217

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18905 BOXES
13 N(W)BOXES
S8 0 S1 AND N(W)BOXES

For 09/732,360

WEST Search History

Yu, Misook

DATE: Tuesday, May 07, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT; PLUR=YES; OP=OR		
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L3	L2 and (screen$ or assay$)	9	L3
L2	L1 and transcript$	9	L2
L1	gabp or hgabp	15	L1

END OF SEARCH HISTORY

09/732,360

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Items	File
281	5: Biosis Previews(R)_1969-2002/May W1
221	34: SciSearch(R) Cited Ref Sci_1990-2002/May W2
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117	71: ELSEVIER BIOBASE_1994-2002/May W1
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15	94: JICST-EPlus_1985-2002/Mar W3
4	98: General Sci Abs/Full-Text_1984-2002/Mar
1	135: NewsRx Weekly Reports_1995-2002/Apr W1
142	144: Pascal_1973-2002/May W1
5	149: TGG Health&Wellness DB(SM)_1976-2002/Apr W4
268	155: MEDLINE(R)_1966-2002/May W1
46	156: ToxFile_1966-2002/Feb W4
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2	172: EMBASE Alert_2002/May W1
11	266: FEDRIP_2002/Mar
5	399: CA SEARCH(R)_1967-2002/UD=13619
1	442: AMA Journals_1982-2002/May B2
2	457: The Lancet_1986-2000/Oct W1
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File 5:Biosis Previews(R) 1969-2002/May W1
(c) 2002 BIOSIS
File 155:MEDLINE(R) 1966-2002/May W1
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File 34:SciSearch(R) Cited Ref Sci 1990-2002/May W2
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File 73:EMBASE 1974-2002/May W1
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see Help News73.

Title: CYCLOSPORINE-A INHIBITS TISSUE FACTOR EXPRESSION IN MONOCYTES/MACROPHAGES

Author(s): HOLSCHERMANN H; DURFELD F; MAUS U; BIERHAUS A; HEIDINGER K; LOHMEYER J; NAWROTH PP; TILLMANNS H; HABERBOSCH W
Corporate Source: UNIV GIESSEN,DEPT INTERNAL MED,DIV CARDIOL,KLINSTR 36/D-35392 GIESSEN//GERMANY/; UNIV GIESSEN,DEPT INTERNAL MED,DIV HEMATOL/D-35392 GIESSEN//GERMANY/; TECH UNIV DRESDEN,FAC MED,INST PATHOL/D-8027 DRESDEN//GERMANY/; UNIV HEIDELBERG,DEPT INTERNAL MED/D-6900 HEIDELBERG//GERMANY/
Journal: BLOOD, 1996, V88, N10 (NOV 15), P3837-3845
ISSN: 0006-4971
Language: ENGLISH Document Type: ARTICLE
Geographic Location: GERMANY
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences; CC CLIN--Current Contents, Clinical Medicine
Journal Subject Category: HEMATOLOGY
Abstract: Accelerated coronary atherosclerosis in cardiac allografts is the major limiting factor for long-term survival after heart transplantation. There is growing evidence that activation of the coagulation mechanism is involved in the development of transplant atherosclerosis. Tissue factor (TF) expression by cells of the monocyte/macrophage system may represent an important mechanism underlying the fibrin deposition in the affected vessels. In the present study, we investigated the **effect** of cyclosporine A (CsA) on the lipopolysaccharide (LPS)-induced procoagulant activity (PCA) in human monocytes/macrophages. CsA exerted a dose-dependent inhibitory **effect** on LPS-induced monocyte/macrophage PCA, which was identified as **TF activity** based on functional and immunologic characterization.

Title: Platelet activating factor causes a rapid increase in activity of prior expressed tissue factor on monocyte surface membrane
Author(s): Oguchi A (REPRINT) ; Morioka M; Obi N; Nishida J; Kinoshita T
Corporate Source: SHIMOINA RED CROSS HOSP,3159-1 MOTO OHJIMA/NAGANO 3993303//JAPAN/ (REPRINT); TEIKYO UNIV,SCH MED, DEPT MED, ITABASHI KU/TOKYO//JAPAN/
Journal: INTERNATIONAL JOURNAL OF HEMATOLOGY, 1999, V69, N2 (FEB), P119-125
ISSN: 0925-5710 Publication date: 19990200
Publisher: CARDEN JENNINGS PUBL CO LTD, BLAKE CTR, STE 200, 1224 W MAIN ST, CHARLOTTESVILLE, VA 22903
Language: English Document Type: ARTICLE
Geographic Location: JAPAN
Subfile: CC CLIN--Current Contents, Clinical Medicine
Journal Subject Category: HEMATOLOGY
Abstract: It is known that tissue factor (TF) activity depends on cell membrane phospholipids. However, the mechanism involved in the regulation of TF activity by the modulation of the phospholipids has not yet been described in detail. To determine whether some mediators regulate TF activity by such a mechanism, we investigated the effect of platelet activating factor (PAF). Addition of PAF to TF-expressed monocytes caused a rapid and marked increase in the activity, but no increase in the antigen. Kinetic analyses were performed on TF-expressed monocytes with or without the addition of PAF, and on purified TF The former revealed that the activity enhancement by PAF was associated with reduced Km, with Vmax remaining unaltered. The latter showed that the additional phosphatidylserine produced greater TF activity in purified TF, with an alteration pattern of kinetic parameters similar to that observed in the addition of PAF From these results, we conclude that PAF regulates TF activity at the cell surface by alteration of the phospholipid composition of the membrane, and not by fresh production of TF apoprotein. The role of PAF as described in this paper must be one of the major regulatory systems in TF activity . (C) 1999 The Japanese Society of Hematology.

Record Date Created: 19980122

7/9/2

DIALOG(R)File 155:MEDLINE(R)

08648192 96064720 PMID: 7592857

Mechanism of the tumor necrosis factor alpha-mediated induction of endothelial tissue factor .

Bierhaus A; Zhang Y; Deng Y; Mackman N; Quehenberger P; Haase M; Luther T ; Muller M; Bohrer H; Greten J; et al

Department of Medicine, University of Heidelberg, Germany.

Journal of biological chemistry (UNITED STATES) Nov 3 1995, 270 (44) p26419-32, ISSN 0021-9258 Journal Code: HIV

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Subfile: INDEX MEDICUS

This study examines the regulation of the human **tissue factor** (TF) **promotor** in vitro and in vivo. Transient transfections were performed in bovine aortic endothelial cells to investigate the role of two fundamentally different AP-1 sites and a closely located NF-kappa B site in the human TF promoter. The NF-kappa B site is functionally active, since overexpression of NF-kappa B(p65) resulted in induction of TF mRNA and activity. Promoter analysis showed that NF-kappa B induction was dependent on the integrity of the region from base pair -188 to -181. Over-expression of Jun/Fos resulted in TF induction of **transcription** and protein/activity. Functional studies revealed that the proximal AP-1 site, but not the distal, was inducible by Jun/Fos heterodimers. The distal AP-1 site, which has a G-->A switch at position 4, was inductible by Jun homodimers. Electrophoretic mobility shift assays, using extracts of tumor necrosis factor alpha (TNF alpha)-stimulated bovine aortic endothelial cells, demonstrated TNF alpha-inducible binding to the proximal AP-1 site, comprising JunD/Fos heterodimers. At the distal AP-1 site, only minor induction of binding activity, characterized as proteins of the Jun and ATF family, was observed. Consistently, this site only marginally participates in TNF alpha induction. Functional studies with TF **promotor** plasmids confirmed that deletion of the proximal AP-1 or the NF-kappa B site decreased TNF alpha-mediated TF induction to a higher extend than loss of the distal AP-1 site. However, integrity of both AP-1 sites and the NF-kappa B site was required for optimal TNF alpha stimulation. The relevance of these in vitro data was confirmed in vivo in a mouse tumor model. Expression plasmids for a dominant negative Jun mutant or I-kappa B were packaged in liposomes. When either mutated Jun or I-kappa B were injected intravenously 48 h before TNF alpha, a reduction in TNF alpha-mediated TF expression in the tumor endothelial cells was observed. Simultaneously, fibrin/fibrinogen deposition decreased and free blood flow could be restored. Thus, TNF alpha-induced up-regulation of endothelial cell TF depends on a concerted action of members of the bZIP and NF-kappa B family.

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: Endothelium, Vascular--metabolism--ME; *Gene Expression --drug effects--DE; *Promoter Regions (Genetics); *Thromboplastin --biosynthesis--BI; *Thromboplastin--genetics--GE; * **Transcription** , Genetic; *Tumor Necrosis Factor--pharmacology--PD; Aorta; Binding Sites; Cattle; Cell Nucleus--metabolism--ME; Cells, Cultured; DNA--chemistry--CH; DNA--metabolism--ME; Kinetics; Mice; NF-kappa B--metabolism--ME; Protein Hybridization; Proto-Oncogene Proteins c-fos--metabolism--ME; Proto-Oncogene Proteins c-jun--metabolism--ME; Recombinant Proteins --biosynthesis--BI; **Transcription** Factor AP-1--metabolism--ME; Transfection

CAS Registry No.: 0 (NF-kappa B); 0 (Proto-Oncogene Proteins c-fos); 0 (Proto-Oncogene Proteins c-jun); 0 (Recombinant Proteins); 0 (Transcription Factor AP-1); 0 (Tumor Necrosis Factor); 9007-49-2 (DNA) ; 9035-58-9 (Thromboplastin)

Record Date Created: 19951221

7/9/3

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Set     Items   Description
S1        307   TISSUE(W)FACTOR AND TRANSCRIP?
S2        131   S1 AND HGABP? OR GABP
S3          0   S1 AND (HGABP OR GABP)
S4       6291   DS
Set     Items   Description
S1        307   TISSUE(W)FACTOR AND TRANSCRIP?
S2        131   S1 AND HGABP? OR GABP
S3          0   S1 AND (HGABP OR GABP)
S4       6291   DS
S5          0   S1 AND (NRF? RO EF? OR E4F1)
S6        159   S1 NOT PY=>1999
S7          3   S1 AND PROMOTOR?
 7/9/1
DIALOG(R)File 155:MEDLINE(R)

09616362   98077369   PMID: 9416887
```

Cyclosporin A inhibits monocyte tissue factor **activation in cardiac transplant recipients.**

Holschermann H; Kohl O; Maus U; Durfeld F; Bierhaus A; Nawroth PP; Lohmeyer J; Tillmanns H; Haberbosch W

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Circulation (UNITED STATES) Dec 16 1997, 96 (12) p4232-8, ISSN 0009-7322 Journal Code: DAW

Languages: ENGLISH

Document type: Clinical Trial; Journal Article

Record type: Completed

Subfile: AIM; INDEX MEDICUS

BACKGROUND: Fibrin deposition and thrombosis have been implicated in both allograft rejection and vasculopathy after cardiac transplantation. Because monocytes play a pivotal role in the pathophysiology of intravascular coagulation activation through their ability to synthesize **tissue factor** (TF), we asked (1) whether monocyte TF activation occurs in cardiac transplant recipients and (2) whether monocyte TF expression is affected by treatment with cyclosporin A (CsA). METHODS AND RESULTS: We measured levels of TF activity in peripheral blood mononuclear cells and highly purified monocytes/macrophages from 10 consecutive cardiac transplant recipients and 10 healthy control subjects. TF activity generated by both unstimulated and endotoxin-stimulated cells was significantly higher in transplant recipients than in control subjects (P<.05). Increased monocyte TF expression in transplant recipients was shown to be adversely affected by treatment with CsA: TF induction was markedly reduced by CsA serum concentrations reaching peak CsA drug levels. Inhibition of TF induction in the presence of high CsA blood concentrations was also observed when stimulation of cells was performed with interferon-gamma or interleukin-1beta. As shown by reverse **transcription** -polymerase chain reaction and electrophoretic mobility shift assay, respectively, treatment with CsA leads to decreased TF mRNA expression and reduced activation of the NF-kappaB **transcription** factor, which is known to contribute to the induction of the TF **promotor** in human monocytes. CONCLUSIONS: This study demonstrates that TF activation, occurring in mononuclear cells of cardiac transplant recipients, is inhibited by treatment with CsA. Inhibition of monocyte TF induction by CsA may contribute to its successful use in cardiac transplant medicine and might be useful in managing further settings of vascular pathology also known to involve TF expression and NF-kappaB activation.

Tags: Female; Human; Male

Descriptors: *Cyclosporine--therapeutic use--TU; *Heart Transplantation; *Monocytes--metabolism--ME; *Postoperative Care; *Thromboplastin --antagonists and inhibitors--AI; Adult; Aged; Middle Age; Monocytes--drug effects--DE; NF-kappa B--metabolism--ME; RNA, Messenger--metabolism--ME; Thromboplastin--genetics--GE; Thromboplastin--physiology--PH

CAS Registry No.: 0 (NF-kappa B); 0 (RNA, Messenger); 59865-13-3 (Cyclosporine); 9035-58-9 (Thromboplastin)

DIALOG(R)File 155:MEDLINE(R)

08507939 95259007 PMID: 7740494

Antiparasitic treatment of patients with P. falciparum malaria reduces the ability of patient serum to induce tissue factor **by decreasing NF-kappa B activation.**

Bierhaus A; Hemmer CJ; Mackman N; Kutob R; Ziegler R; Dietrich M; Nawroth PP

Dept. of Medicine I, Univ. of Heidelberg, Germany.

Thrombosis and haemostasis (GERMANY) Jan 1995, 73 (1) p39-48, ISSN 0340-6245 Journal Code: VQ7

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Subfile: INDEX MEDICUS

Serum from patients with P. falciparum malaria at day 1 (pretherapy) induces **tissue factor** (TF) in cultured endothelial cells. TF induction depends on de novo **transcription** as shown in Nuclear Run On assays. Electrophoretic mobility shift assays demonstrated binding of AP-1 and NF-kappa B/Rel proteins to their recognition sites in the TF **promotor** . After therapy (day 28), stimulation of TF antigen by patient serum is reduced by 70%. When serum obtained before and after therapy was compared, a decrease of NF-kappa B activation was evident. Activation of NF-kappa B-like proteins was in part dependent on TNF alpha in patient serum, since a TNF alpha neutralizing antibody reduced induction of TF **transcription** and translation and induction of NF-kappa B-like proteins. Induction of TF activity was suppressed by pDTC, an inhibitor of NF-kappa B activation. When different **promotor** constructs of the TF gene were tested, induction was dependent upon the presence of the intact NF-kappa B-like binding site in the TF **promotor** . A mutant with deleted NF-kappa B, but intact AP-1 sites was not inducible. Mutation of the AP-1 sites did not prevent induction, but reduced inducibility by pretherapy serum. Therefore, NF-kappa B/Rel proteins are responsible for induction of TF **transcription** by pretherapy serum, but AP-1 is needed for highest inducibility. The effect of antiparasitic therapy on the induction of TF by serum from patients with complicated P. falciparum malaria is dependent on a therapy-mediated loss of activation of NF-kappa B-like proteins in post-treatment patient serum.

Tags: Comparative Study; Human; Support, Non-U.S. Gov't

Descriptors: *Antimalarials--pharmacology--PD; *Gene Expression Regulation--drug effects--DE; *Malaria, Falciparum--blood--BL; *NF-kappa B --antagonists and inhibitors--AI; *Thromboplastin--biosynthesis--BI; *Tumor Necrosis Factor--physiology--PH; Antimalarials--therapeutic use--TU; Base Sequence; Binding Sites; Cells, Cultured; Endothelium, Vascular--drug effects--DE; Endothelium, Vascular--metabolism--ME; Genes, Reporter; Malaria, Falciparum--drug therapy--DT; Molecular Sequence Data; Mutagenesis ; NF-kappa B--physiology--PH; Promoter Regions (Genetics); Recombinant Fusion Proteins--biosynthesis--BI; Recombinant Fusion Proteins--genetics --GE; Regulatory Sequences, Nucleic Acid; Thromboplastin--genetics--GE; **Transcription** Factor AP-1--metabolism--ME; **Transcription** , Genetic--drug effects--DE; Transfection; Tumor Necrosis Factor--analysis--AN; Umbilical Veins

CAS Registry No.: 0 (Antimalarials); 0 (NF-kappa B); 0 (Recombinant Fusion Proteins); 0 (Transcription Factor AP-1); 0 (Tumor Necrosis Factor); 9035-58-9 (Thromboplastin)

Record Date Created: 19950605

As shown by reverse transcriptase-polymerase chain reaction, CsA reduced the transcription of the TF gene in LPS-stimulated monocytes/macrophages. Electrophoretic mobility shift assay showed that CsA inhibited the LPS-induced activation of the nuclear factor KB (NF-KB), AS shown by Western blot analysis, CsA treatment decreased the nuclear translocation of NF-KB, thereby suggesting the mechanism for the inhibitory **effect** of CsA on TF induction. Hence, a nonimmunologic **effect** of CsA may contribute to its successful use in transplant medicine. (C) 1996 by The American Society of Hematology.

Set	Items	Description
S1	1035	TF(W)ACTIVIT?
S2	696	S1 NOT PY=>1999
S3	0	S1 AND GABP
S4	553	S1 AND (NRF? OR E4F? OR EF?)
S5	7	S4 AND VIRUS

5/9/3 (Item 3 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03567180 Genuine Article#: PN490 Number of References: 33
Title: EFFECTS OF PROSTACYCLIN ANALOGS ON THE SYNTHESIS OF TISSUE FACTOR, TUMOR-NECROSIS-FACTOR-ALPHA AND INTERLEUKIN-1-BETA IN HUMAN MONOCYTIC THP-1 CELLS
Author(s): CRUTCHLEY DJ; CONANAN LB; QUE BG
Corporate Source: MIAMI HEART RES INST,4701 MERIDIAN AVE/MIAMI BEACH//FL/33140
Journal: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, 1994, V271, N1 (OCT), P446-451
ISSN: 0022-3565
Language: ENGLISH Document Type: ARTICLE
Geographic Location: USA
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: PHARMACOLOGY & PHARMACY
Abstract: Previous studies have shown that prostacyclin analogs can inhibit the expression of tissue factor (TF) procoagulant activity by human monocytes. The present studies have investigated this phenomenon further, by using a plasma coagulation assay to measure cellular **TF activity**, an immunoassay to measure TF antigen and reverse transcription/polymerase chain reaction with appropriate oligomer primers to measure if mRNA. Iloprost and cicaprost inhibited lipopolysaccharide-induced increases in **TF activity**, antigen and mRNA (50% inhibition, 2-8 nM), with no apparent **effect** on TF mRNA stability. These agents therefore act at or before the level of transcription of the TF gene. The analogs were more potent inhibitors of tumor necrosis factor-alpha synthesis (50% inhibition at 334 +/- 40 pM cicaprost or 846 +/- 182 pM iloprost) and extraordinarily potent when combined with a phosphodiesterase inhibitor (50% inhibition at 101 +/- 31 pM iloprost in the presence of 20 mu M isobutylmethylxanthine). Iloprost and cicaprost were less potent in inhibiting the synthesis of interleukin-1 beta (50% inhibition, 50-100 nM). Cicaprost inhibited lipopolysaccharide-induced increases in mRNA levels for TF, tumor necrosis factor-alpha and interleukin-1 beta; differential potency was again observed. We conclude that these three important monocyte functions can be down-regulated by prostacyclin analogs, and with differential sensitivity. Furthermore, the extreme sensitivity of tumor necrosis factor-alpha synthesis to inhibition suggests that such inhibition may be a major physiological function of prostacyclin

5/9/5 (Item 5 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03066145 Genuine Article#: NB787 Number of References: 20
Title: THE EFFECT OF CALCIUM IONOPHORE A23187 ON TISSUE FACTOR ACTIVITY AND MESSENGER-RNA IN ENDOTHELIAL-CELLS
Author(s): WAKITA K; STEARNSKUROSAWA DJ; MARUMOTO Y

Corporate Source: DAIICHI PHARMACEUT CO LTD,DEPT MOLEC BIOL RES LAB,16-13 KITAKASAI 1 CHOME,EDOGAWA KU/TOKYO 134//JAPAN/; DAIICHI PHARMACEUT CO LTD,DEPT MOLEC BIOL RES LAB,16-13 KITAKASAI 1 CHOME,EDOGAWA KU/TOKYO 134//JAPAN/
Journal: THROMBOSIS RESEARCH, 1994, V74, N2 (APR 15), P95-103
ISSN: 0049-3848
Language: ENGLISH Document Type: ARTICLE
Geographic Location: JAPAN
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: HEMATOLOGY; CARDIOVASCULAR SYSTEM
Abstract: Tissue factor (TF) is an integral membrane glycoprotein that serves as a cofactor for blood coagulation factor VIIa. The induction of TF on the surface of endothelial cells is initiated by various kinds of stimuli including lipopolysaccharide (LPS), interleukin-1beta (IL-1beta), and tumor necrosis factor alpha (TNFalpha). The mechanisms leading to induction of TF are largely un-known and the present study explores the influence of calcium influx on TF induction in LPS-stimulated human umbilical vein endothelial cells. TF cofactor activity was measured on cell surfaces and in lysates by a two-stage chromogenic assay after the cells were incubated under a variety of conditions. **TF activity** of cell surfaces increased 3.3-fold above control values after LPS stimulation (100 ng/ml, 4 h). Addition of 20 muM A23187, a calcium ionophore, to the LPS-stimulated cells just before the TF assay, resulted in an additional 8.8-fold enhancement. **TF activity** of lysed cells increased 10.5-fold above control values after LPS stimulation (100 ng/ml). Incubation with lower concentrations of A23187 and 100 ng/ml-1 mug/ml LPS for 4 h resulted in activity twice that of LPS stimulation alone. The TF mRNA signal of LPS plus 1 muM A23187-treated cells was also increased in addition to LPS treated cells.